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Section on Nervous and Mental Diseases, American Medical Association

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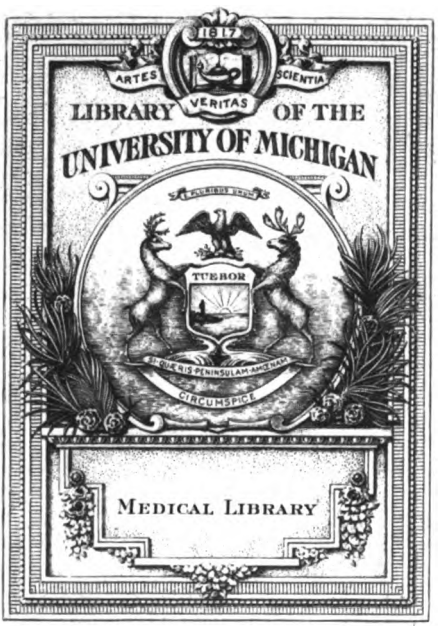

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TRANSACTIONS OF THE

SECTION ON

Nervous and Mental Diseases

of the
American Medical Association
at the Sixty-First Annual
Session, held in St. Louis,
Mo., June 7 to 10, 1910

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LIST OF OFFICERS

This Section has been in existence under its present name since 1900. The branches of the practice of medicine which comprise its province were classified with some other subjects in the early days, as is shown by the following section names. The list of officers has been compiled from the published records, which are not complete—for example, the city is not given in some cases. It will be appreciated if any additional information is brought to the attention of the American Medical Association.

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CONTENTS

PAGE

Proceedings of the Section 11

Chairman's Address: The Institutional Treatment of Nervous and Mental Diseases. Theodore Diller .. 15

The Dependence of Neurology on Internal Medicine. A Plea for the Establishment of Neurologic Hospitals, and of Neurologic Wards in General Hospitals. Joseph Collins and Pearce Bailey 25

The State Care of the Inebriate. Irwin H. Neff 34

The Topographic Diagnosis of Subtentorial Brain Tumors. Herman H. Hoppe 50

Brain Tumor of Psychomotor Area, Causing Jacksonian and Generalized Convulsions, Visual Hallucinations, Somatic Delusions, Astereognosis and Hemiplegic Paralysis. Operation. Recovery, Mental and Physical. Frank W. Langdon and Simon Pendleton Kramer 76

Three Cases of Tumor in the Cerebello-Pontile Angle. Julius Grinker 81

The Treatment of Acute Ailments in Persons Addicted to the Habitual Use of Narcotics. George E. Pettey 99

Manual Work in the Treatment of the Functional Nervous Diseases. Herbert J. Hall 108

Gymnastics and Massage in the Treatment of Nervous Diseases. Haldor Sneve 115

Nervous and Mental Disturbances of the Male Climacteric. Archibald Church 129

Amyotonia Congenita. The Report of a Case With Muscle Biopsy. A. L. Skoog 137

The Relation of Stuttering to Amusia. E. Bosworth McCready 148

Choked Disc in its Relation to Cerebral Tumor and Trephining. Alfred Saenger 155

Pathology and Bacteriology of Acute Anterior Poliomyelitis. H. E. Robertson and A. J. Chesley 170

The Surgical Treatment of Infantile Spinal Paralysis. David Silver 196

Digitized by Google

CONTENTS

PAGE.

Intradural Tumor of the Spinal Cord with Operation. Edward E. Mayer and Otto C. Gaub 222

Hemiplegia Following Acute Infections. W. A. Jones and Arthur S. Hamilton 235

Syphilitic Disease of the Arteries of the Central Nervous System, With Detailed Report of a Case. E. M. Hummel 252

The Hemianoptic Prism-Phenomenon of Wilbrand. Alfred Saenger 261

Index 265

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PROCEEDINGS OF THE SESSION

TUESDAY, JUNE 7—MORNING

The meeting was called to order at 2 p. m., in the St. Louis University Library, by the Chairman, Dr. Theodore Diller, Pittsburg, who then read his address.

A committee, consisting of Dr. Collins and Dr. Langdon, was appointed to prepare resolutions regarding the death of Dr. Wharton Sinkler.

Dr. Alfred Saenger, of Hamburg, Germany, was, on motion, recommended for honorary membership. [The House of Delegates approved.]

A resolution deprecating the custom of printing details of suicides in the secular press on account of the harmful, suggestive effect on certain individuals, and requesting that the Legislative Committee be instructed to secure, if possible, the enactment of a national law forbidding the use of the mails to newspapers publishing such details, and making it a misdemeanor to publish them, was referred to the Executive Committee.

Dr. Joseph Collins, New York, read a paper on "The Urgent Need of Hospitals for the Study and Treatment of Nervous Diseases."

Dr. L. Vernon Briggs, Boston, read a paper on "Prevention of Insanity by the Treatment of Incipient Cases in General Hospitals."

Dr. Irwin H. Neff, Foxboro, Mass., read a paper on "The State Care of the Inebriate."

These three papers were discussed by Drs. Alfred Saenger, Hamburg, Germany; Charles L. Dana, New York; T. D. Crothers, Hartford, Conn.; F. P. Norbury, Kankakee, Ill.; A. E. Sterne, Indianapolis; Frank R. Fry, St. Louis; Philip Zenner, Cincinnati; Hugh T. Patrick, Chicago; Theodore Diller, Pittsburg; Haldor Sneve, St. Paul, Minn.; Herman H. Hoppe, Cincinnati; Joseph Collins, New York; L. Vernon Briggs, New York; and Irwin H. Neff, Foxboro, Mass.

The Chairman appointed as an Executive Committee, and also as a Committee on Nomination of Officers: Drs. Charles L. Dana, New York; H. T. Patrick, Chicago; and F. W. Langdon, Cincinnati.

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Dr. Herbert J. Hall, Marblehead, Mass., read a paper on "Manual Work in the Treatment of the Functional Nervous Diseases."

Dr. Haldor Sneve, St. Paul, Minn., read a paper on "The Treatment of Nervous Diseases by Gymnastics and Massage."

These two papers were discussed by Drs. C. R. Woodson, St. Joseph, Mo.; John Punton, Kansas City, Mo.; Frank R. Fry, St. Louis; Philip Zenner, Cincinnati; C. P. Emerson, Clifton Springs, N. Y.; L. H. Mettler, Chicago; Theodore Diller, Pittsburg, Pa.; Herbert J. Hall, Marblehead, Mass.; and Haldor Sneve, St. Paul, Minn.

Wednesday, June 8—Morning

Dr. H. E. Robertson, Minneapolis, read a paper on "Pathology of Anterior Poliomyelitis."

Dr. David Silver, Pittsburg, Pa., read a paper on "The Surgical Treatment of Anterior Poliomyelitis."

These two papers were discussed by Drs. Archibald Church, Chicago; L. H. Mettler, Chicago; P. Bassoe, Chicago; A. L. Skoog, Kansas City; Arthur S. Hamilton, Minneapolis; Nathaniel Allison, St. Louis; Frank R. Fry, St. Louis; C. R. Woodson, St. Joseph, Mo.; C. H. Hughes, St. Louis; H. E. Robertson, Minneapolis; and David Silver, Pittsburg.

Wednesday, June 8—Afternoon

Dr. Herman H. Hoppe, Cincinnati, read a paper on "The Topographical Diagnosis of Subtentorial Tumors."

Dr. Frank Warren Langdon and Simon P. Kramer, Cincinnati, presented a paper on "Brain Tumor of the Psychomotor Area."

Dr. Julius Grinker, Chicago, read a paper on "Tumor of the Cerebello-Pontile Angle."

These three papers were discussed by Drs. C. H. Hughes, St. Louis; George W. Hall, Chicago; A. E. Sterne, Indianapolis; Charles L. Dana, New York; Theodore Diller, Pittsburg, Pa.; D'Orsay Hecht, Chicago; H. H. Hoppe, Cincinnati; and Julius Grinker, Chicago.

Drs. Edward E. Mayer and Otto C. Gaub, Pittsburg, Pa., presented a paper on "Intradural Tumor of Lower Cervical Cord with Operation."

Discussed by Drs. Theodore Diller, Pittsburg, Pa.; Julius Grinker, Chicago; Charles L. Dana, New York; and Edward E. Mayer, Pittsburg, Pa.

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Dr. Archibald Church, Chicago, read a paper on "Nervous and Mental Disturbance at the Male Climacteric." No discussion.

Dr. Charles L. Dana, New York, read a paper on "Study and Interpretation of Pains and Dysthesias." Discussed by Drs. Frank R. Fry, St. Louis; John Punton, Kansas City, Mo.; Sanger Brown, Chicago; L. H. Mettler, Chicago; Julius Grinker, Chicago; C. A. Wingerter, Wheeling, W. Va.; William W. Graves, St. Louis; Theodore Diller, Pittsburg; Charles L. Dana, New York.

The following statement was adopted by rising vote in memory of the late Wharton Sinkler:

The Section on Nervous and Mental Diseases of The American Medical Association desires to record the death of one of its most distinguished and faithful members, a former chairman, and a loyal supporter, Dr. Wharton Sinkler of Philadelphia.

Dr. Sinkler achieved eminence in his profession and affection in the hearts of his colleagues. To him his profession was an ideal, and to his professional brethren he was ideal. Sane, honest, courageous and industrious, blessed with a sense of humor, and richly endowed with a fund of sympathy, his presence was invariably a source of pleasure to his friends and a tower of strength to his patients.

To us his loss is irreparable. We mourn him, and we shall continue to do so, but not so long as we shall praise him.

JOSEPH COLLINS and F. W. LANGDON, Committee.

Dr. E. M. Hummel, New Orleans, read a paper on "Syphilitic Disease of Arteries of the Central Nervous System, with Detailed Report of a Case." Discussed by Drs. William W. Graves, St. Louis; George A. Moleen, Denver; Sanger Brown, Chicago; Theodore Diller, Pittsburg, and E. M. Hummel, New York.

Dr. A. L. Skoog, Kansas City, Mo., read a paper on "Amyotonia Congenita." Discussed by Dr. Theodore Diller, Pittsburg, Pa.

Dr. E. Bosworth McCready, Pittsburg, Pa., read a paper on "The Relation of Stuttering to Amusia." No discussion.

THURSDAY, JUNE 9—MORNING

The following officers of the Section for 1911 were elected: Chairman, W. A. Jones, Minneapolis; Vice-Chairman, Herman H. Hoppe, Cincinnati; Secretary, E. E. Southard, Boston; Delegate, Theodore Diller, Pittsburg, Pa.; Alternate, J. H. McBride, Pasadena, Cal.

The resolution deprecating the custom of printing details of suicides was adopted as the sense of the Section, and this

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action was ordered referred to the House of Delegates of the American Medical Association for action. That body modified the resolution and adopted it in the following form:

Resolved, That it is the sense of the House of Delegates of the American Medical Association that the publication of the details of suicide by newspapers is one of the potent causes of other suicides by the suggestive influence of such published details; and it is, therefore, recommended to the public press that the details of suicide be omitted from publication.

Dr. Alfred Saenger, Hamburg, Germany, read an address on "Choked Disc in Its Relation to Cerebral Tumor and Trepanning." Discussed by Drs. J. Herbert Parsons, London; Julius Grinker, Chicago; J. Bordley, Jr., Baltimore; N. M. Black, Milwaukee, Wis., and Alfred Saenger, Hamburg, Germany.

Dr. Alfred Saenger, Hamburg, Germany, illustrated and discussed Wilbrand's prism test.

Dr. W. W. Graves presented two cases, one of brain tumor and another of a condition of hemianopsia, etc., resulting from skull fracture. Discussed by Dr. Alfred Saenger, Hamburg, Germany.

Dr. John Punton, Kansas City, Mo., read a paper on "The Plea of Insanity as a Defense for Crime and Its Remedy." Discussed by Drs. Louise G. Robinovitch, New York; David S. Booth, St. Louis; A. E. Sterne, Indianapolis; George A. Moleen, Denver; C. W. Thierny, St. Louis; L. V. Briggs, Boston; C. A. Wingerter, Wheeling, W. Va.; C. R. Woodson, St. Joseph, Mo.; Edward Bow, Jacksonville, Ill.; John Punton, Kansas City, Mo.; S. G. Burnett, Kansas City, Mo., and Sanger Brown, Chicago.

Drs. W. A. Jones and Arthur S. Hamilton, Minneapolis, presented a paper on "Hemiplegia Following Acute Infections." Discussed by Drs. M. M. Perry, Parsons, Kan.; Sanger Brown, Chicago; L. H. Mettler, Chicago, and W. A. Jones, Minneapolis.

Dr. George E. Pettey, Memphis, Tenn., read a paper on "The Treatment of Acute Ailments Occurring in Persons Addicted to Narcotic Drugs." No discussion.

Dr. George Edward Fell, Buffalo, N. Y., read a paper on "Twenty Years of Electrocution." No discussion.

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THE INSTITUTIONAL TREATMENT OF NERVOUS AND MENTAL DISEASES

CHAIRMAN'S ADDRESS

THEODORE DILLER, M.D.
Clinical Professor of Neurology, University of Pittsburg
PITTSBURG, PA.

It is my pleasant duty in opening the proceedings of this Section to thank you for the honor you have bestowed on me in making me the Chairman of the Section for the year. I may further say that the honor is a real and unexpected one; for the first intimation which I had that I was to be chairman for the year was the report of the nominating committee to the Section at the meeting of the Association last year at Atlantic City. And this leads me to believe that, whatever may be said of the past, or of any other Section of this Association, in the Section on Nervous and Mental Diseases, medical politics plays no part whatever.

I believe that it should be the aim of this Section to discuss all the varied phases of nervous and mental diseases, and that its officers should endeavor to assemble together the best available men in the country on its program. And I further believe that it should be the especial aim of this Section to present the subjects of neurology and psychiatry in such clear, simple and attractive manner that its proceedings may serve to interest and instruct not only neurologists and alienists, but also general practitioners and internists and a certain number of those whose field of activity lies in work in other specialties. It may be truthfully said that the problems which engage the interest of the members of

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this Section also possess in themselves more or less interest for all members of the profession. Neurology and psychiatry in their several aspects and in their manifold interests call on those who pursue them as specialties to take more or less account of every other department in the broad domain of medicine and, indeed, much outside it, for example, law, social science and penology.

It is with this understanding of the subject that I have endeavored to cooperate with our secretary (and to work with him has been a genuine pleasure) in making out the program for this meeting.

The criticism is often made of programs of associations of neurologists and alienists that they present little or nothing on the subject of treatment; and for one, I have the feeling that this reproach is not wholly undeserved. Hence it has been our aim in making up the program for the Section this year to secure an unusual number of good and practical papers dealing with the treatment of nervous and mental diseases; and I believe that if you will look over the program in your hands you will agree with me that we have attained a very fair measure of success.

I wish to address the Section briefly on a subject which I believe to be one of vast and far-reaching importance, even though I shall be able to speak only in general terms and must treat it most inadequately and leave much that I should say unsaid. I refer to the institutional treatment of nervous and mental diseases.

This subject becomes one of vast importance when we consider the large number of patients for the adequate or proper care of whom institutional treatment is required. In nearly all cases of insanity institutional treatment is demanded and received; in nearly all the severer cases of inebriety institutional treatment is demanded, but in few is it received; and very many of the cases of organic and functional nervous diseases demand and some receive institutional care.

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As years go by and hospitals for the insane improve their equipment, and as the public becomes better educated as to the value of institutional treatment for the insane and less and less suspicious of these institutions, the percentage of insane and feeble-minded outside of hospitals grows less and less, and these institutions everywhere throughout the broad land are becoming more crowded. Nor does the provision for additional accommodations in any state in the Union keep pace with the growing demands for such accommodations. Despite many drawbacks and obstacles, and despite the great work of improvement which yet remains to be accomplished, these institutions have made fair progress in their methods and equipment for the care and treatment of the insane and feeble-minded.

In this connection I desire to direct attention to the subject of the treatment of insane in institutions other than regular hospitals or asylums for the insane. So far as I know, there are only two general hospitals in the United States—St. Francis Hospital, Pittsburg, and Albany Hospital, Albany, N. Y.—which are equipped to receive insane patients. There are several large hospitals for the insane which are departments of general hospitals, but which possess separate buildings and separate managements and must be regarded as practically separate institutions.

I wish to say a word here regarding St. Francis Hospital in Pittsburg and its work to illustrate some of the points I wish to make. This is a large general hospital and has connected with it a psychopathic ward. In this ward or department there are about 100 patients, including a great many acute cases; and it has been the aim to have only acute cases in this department. A resident physician and intern devote all their time to this department, which is under the medical direction of three visiting physicians. The members of the medical and surgical staff are frequently conferred with regarding these patients. While

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the psychopathic department of St. Francis Hospital is not entirely ideal and much remains to be done to make it so, yet it stands for a most valuable principle, namely, the treatment of insane in a general hospital. This means much, very much. Our experience at St. Francis is that patients suffering from acute psychoses are admitted early, and that their friends do not feel that the same stigmata is placed on them going to and coming from this hospital as in the case of the regular hospital for insane. It is well known that certain psychoses can be and have been treated in general hospitals, but the difficulty is that if such patients become noisy or troublesome the management generally demands their removal. I am strong in the conviction that in every large city there should be at least one general hospital ready to receive cases of insanity and equipped to treat these patients. A large general hospital can easily have a properly equipped wing or even detached section to hold 25 or 50 patients, and thus it can accomplish a great purpose and fill a great need in any large community. The noise and disturbance of such patients can be kept from the general hospital if the wing or separate building is properly planned and built.

Psychiatry should be regarded as a department of internal medicine, and I trust that the time will come when the psychoses will be discussed in general textbooks in the practice of medicine. And the day is coming also, I trust, when patients with acute psychoses will receive treatment in general hospitals and not have to go to hospitals for the insane unless the disease is unduly prolonged or appears to be incurable. When the treatment of insanity is undertaken by general hospitals, as I have indicated, I believe that an entire change will come over the profession and the public regarding their attitude toward the insane. The profession will realize in a practical way that what is called insanity is an expression of sickness; and the laity will learn the same lesson. Another result would be that

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the skill of the various specialties would be brought to the insane; and these patients would be a constant source of education for all physicians connected with hospitals maintaining such psychopathic departments. And then psychoses would be described in the text-books on the practice of medicine and the general profession would learn something about them.

It appears to me that, viewed from almost any aspect, the treatment of insanity in general hospitals is greatly to be desired and promoted. Nor do I believe that separate psychopathic hospitals, isolated and apart from the general hospital, possess the advantage which a psychopathic division of a general hospital possesses; for sooner or later they are sure to be regarded by the general public as "insane asylums."

There is a large and sadly growing class of individuals for whom large provision should be made for institutional treatment and for whom such provision is now sadly lacking if we are to hope to give anything like adequate treatment; I refer to the inebriate, addicted to the excessive and uncontrolled or uncontrollable use of alcoholic drinks or morphin, cocain or other drugs. The present inadequate treatment of inebriety is to-day, in my opinion, a standing reproach to society and to the medical profession. The inebriate who seeks or whose family seeks for him institutional treatment meets at all hands with invitations from quackish institutions whose treatment in the main is simple humbug. And yet I think that it will be generally admitted that for a large number of inebriates there can be no adequate treatment, indeed none worth the name, other than that afforded by a well-equipped institution. And such institutions are few in number; so few, indeed, that many an honest practitioner knows not where to find even one for his inebriate patient.

While there is and will be an increasing number of private institutions for inebriates, these are and must be wholly inadequate to meet the needs of the situation.

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As the result of much thought and investigation, I have reached the conclusion that the several states must provide separate institutions for inebriates, and such institutions should be governed by clear, adequate and comprehensive laws.

Massachusetts and Iowa are, so far as I know, the only states which at present maintain state hospitals for inebriates; and both have adequate laws governing them. We are fortunate in having with us at this meeting Dr. Neff, the superintendent of the Massachusetts State Hospital for Inebriates, who is to tell us of the workings of that institution. Minnesota is about to provide a state institution for inebriates, and the subject has been urged before the New York and New Jersey legislatures, but as yet without success.

In Pennsylvania, the state medical society has for several years been trying to persuade the legislature to build a state hospital for inebriates. A bill providing an appropriation for the creation of such an institution was passed by the last assembly; but it was vetoed by the governor because of his belief that the financial situation would not warrant this new expenditure. Pennsylvania already has a law which provides that an inebriate may be committed by a judge, after a hearing, to a "proper hospital or asylum" for a period not to exceed one year. Under this law, a number of inebriates have been committed to various hospitals for the insane throughout the state. Superintendents of these institutions are unanimous in saying that they do not desire these patients, as they do not properly belong there, that they are hard to get on with and upset the discipline of the institution. On the other hand, the inebriate himself strongly resents being committed to an insane asylum; and in consequence he goes there, when compelled to, in a frame of mind little calculated to effect a restoration to health having in it the elements of stability.

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Our argument to the Pennsylvania legislature has been brief and to the effect that the adequate treatment of inebriety requires a separate and properly equipped institution and a proper law governing it and the inebriate. It was urged that, since the state has taken the first step in providing a law governing inebriates, it should logically take the second step and provide the institution which the law contemplates. We hope next winter to succeed in our endeavors with the legislature.

Thus it will be seen that already a start has been made in the matter of provision of state institutions for inebriates and I trust that in the near future we shall see the establishment of many such institutions, so that ultimately every state shall possess at least one.

And now, finally, I shall consider one other class of institutions, namely, sanitariums having for their object the care and treatment of persons suffering from organic and functional nervous disease. These institutions are of all sorts, kinds and descriptions and they are found in all parts of the country. They are large and small; meagerly equipped and elaborately equipped; they are operated by religious associations and by commercial companies and by individuals; they are ethical and semi-ethical and quackish in character. And after a large experience with them, covering something like a score of years, I am bound to say that, generally speaking, sanitarium treatment in this country leaves much, very much, to be desired. My objections to sanitariums are various. This one is entirely too large and has too many patients and too large and complicated machinery and possesses the "sanitarium atmosphere" which is oppressive and depressive. Another is small and badly equipped and affords too little diversion; treatment is slovenly and routine. It appears to me that it is perfectly proper and desirable that sanitariums should afford an opportunity for religious devotions, but it also seems highly objectionable to me that a religious atmosphere should be forced or obtruded on patients. Again, we

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find that some articles of diet are recommended and others prohibited which are in ordinary use, and thus patients run a great risk of becoming diet cranks. We see sometimes an elaborately equipped sanitarium with all sorts of apparatus, all of which looks very impressive to the casual visitor, but which is too often used improperly. It is used improperly, I believe, when used unnecessarily or too frequently, or when the ends to be gained are not properly considered. I have known more than one self-centered, introspective patient to be made worse by the constant round of ill-planned or unplanned examinations and "treatments" in sanitariums. Again, there may be an excellent medical man at the head of a sanitarium; but the physician finds that when this patient goes to the institution he did not see this excellent chief, but an underpaid and inadequate subordinate. Generally speaking, sanitariums do not sufficiently provide for outdoor games, exercises, walks and varied amusements; and the treatment is slovenly and too mechanical and routine.

Instead of going further into the matter of my objections to sanitariums as I have found them, I shall endeavor to make a little sketch of the ideal sanitarium as I see it in my mind's eye, and as one often finds it abroad; and as one very, very seldom sees it in this country.

It is not very large; it accommodates from 50 to 100 patients. It is situated in the country about three-quarters of a mile from the railroad and is reached by a charming drive; it is in the midst of a very charming landscape in which are found no objectionable features. A large wood in the neighborhood belongs to the sanitarium and has many paths and walks running through it, and these are marked off so that patients can estimate the distance they have walked. Scattered along these walks at intervals are various benches and a few pavilions.

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A stable is attached to the sanitarium; drives in the neighborhood are attractive and varied. In a separate building, but very close to the sanitarium and connected with it by a passage way, is an amusement hall containing auditorium for private theatricals and concerts, etc., and in the basement there are billiard tables and a bowling alley and a swimming pool, all of which are used every day. Closely adjoining the amusement hall there is a small but well-equipped bathing establishment with Turkish baths and other provisions for hydrotherapy. Those taking Turkish baths have easy access to the swimming pool.

Now as to the sanitarium itself. It has been especially built and equipped for sanitarium purposes. The building is modest and attractive and entirely free from any freakish design. It has a home-like appearance. The furnishings throughout are simple and modest and thoroughly good, and the whole place throughout is absolutely clean. Nothing is obtrusive. The physician in charge is a man well known for his excellent general character, his ethical standing and his sound conservatism, and the fact that he keeps in touch and abreast with the medical world of the day. In short, he is a man of experience and soundness of character and he possesses tact in a high degree so that he gets on well with the various patients. His position is secure and his time is wholly devoted to his medical duties. He never hesitates to recommend the dismissal of a patient when such dismissal seems to be for the best interests of the patient or the institution. When patients become insane or unmanageable he has backbone enough to demand their dismissal and that they be placed in a proper institution.

The physician in chief is assisted by one or two younger medical men who breathe the atmosphere of their chief and work in complete harmony with him.

The various apparatuses for laboratory work and investigation and therapeutic purposes are at hand. These

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are used with caution and discretion and not in blind routine fashion and seldom needlessly. One of the main features of the sanitarium is the stress laid on individual treatment; and the doctor avoids, so far as possible, cut-and-dried routine in all his examinations and treatments. The rules governing the sanitarium are simple and clear. Nothing is obtrusive or "peculiar" in these rules regarding religion, diet or anything else. The dietary is simple, but sufficiently varied and thoroughly good. A short religious service is held every day, and any one can go to it; it is held in a place apart and is not in any way obtruded on those who do not wish to go and no one is ever pressed to go.

This is to my mind, as nearly as I can picture it for myself, the ideal sanitarium. The rates are a little high; but not higher than they ought to be considering what the patient gets. And whether or not you accept this ideal sanitarium, I believe that there is to-day a great need and growing demand for better sanitarium treatment in this country.

Westinghouse Building.

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THE DEPENDENCE OF NEUROLOGY ON INTERNAL MEDICINE

A PLEA FOR THE ESTABLISHMENT OF NEUROLOGIC HOSPITALS, AND OF NEUROLOGIC WARDS IN GENERAL HOSPITALS

JOSEPH COLLINS, M.D.

AND

PEARCE BAILEY, M.D.

Physicians to the Neurological Institute of New York

NEW YORK

The practice of establishing institutions for special research, with endowments sufficient to secure them from the motives which influence the conduct of establishments on a commercial foundation, is rapidly gaining substantial recognition in America. We have had no war indemnities applicable to scientific purposes, but fortunately we have scores of wealthy men and women who are showing themselves disposed to lay those financial foundations which must henceforth underly the upbuilding of the natural sciences. They have initiated a movement which has already done more for us than herald a coming greatness. They have stimulated interest in medical progress, both in the profession and out of it, and already practical results are at hand.

NEUROLOGY NEGLECTED

Our achievements in surgery, in medicine, in public hygiene, in the widening scope of medical education, are all products of the spirit of inquiry which is fostered in the endowed laboratory; and such laboratories are springing up everywhere. But while the land, throughout its length and breadth, is preparing, as it were, for

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the responsibilities of leadership, one branch of medicine has been accorded no opportunity of sharing in the general progress. This branch is neurology. There seems to have been no thought of providing for its future, and its place in America is about what it was twenty-five years ago. Neither laboratories nor hospitals are being endowed in its favor. In all America there is scarcely a general hospital with neurologic wards worthy of the name, with the exception of a few hospitals and inaccessible city almshouses in which cripples and dotards are crowded together under the care of untrained housestaffs without laboratory facilities. Magnificent endowments almost yearly further the advances in surgery, medicine, and psychiatry, but neurology is left to take care of itself. Few nurses receive special training in nervous diseases; the objective teaching of students is limited chiefly to ambulatory cases. The profession who teach them, having no hospital patients of their own, can demonstrate only such patients as are able to walk to the clinic. In the recognition of brain abscess, meningitis, acute intoxications and injuries of the nervous system, the student either goes uninstructed, or else gets his instruction from men neither particularly interested nor particularly versed in these subjects. Hospital section-teaching, the most valuable of all modern teaching methods, is practically unknown in nervous diseases. The clinical facilities of the professors of neurology are not much better than those of the students. Without wards, without laboratories, without means for the continuous daily observations of patients, the holders of these chairs cannot obtain the intimate knowledge of disease, and the stimulus to investigate it, which is necessary to keep them abreast of modern progress.

In most large cities the general hospitals appoint what is known as a consulting neurologist. This incumbent may or may not be a member of the medical board. He is admitted to the wards by invitation and on sufferance; and such advice as he gives may be acted

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upon or not, in accordance with the whim or acumen of the attending physician or surgeon. No house officer is accredited to him, and he can follow a case only by extreme effort, and, when all is over, he is permitted to utilize it for scientific purposes only in the event of the attending physician or surgeon not wishing to make use of it himself. He is excluded from the consecutive observation and the scientific study of the metabolic diseases which underlie so many functional nervous affections.

NEUROLOGY AS A SPECIALTY

One cannot soberly consider these facts without being confronted with two important questions: Is neurology a legitimate specialty? If it is, what must be done, not only to advance it, but to enable it to hold its place? The answer to the first question is certainly affirmative. Ever since the publication of Erichsen's book on spinal injuries, it has become every year more obvious that nervous diseases require men of special equipment, judgment and inclination to interpret them correctly. It is more special in study and practice than any of the recognized subdivisions of surgery; just as special and far more extended than opthalmology or otology. So thoroughly has it been recognized as a field of activity *sui generis* that no argument on the subject seems necessary. Its individuality being granted, the next question is, Where shall we turn for successors to Duchenne, Lockhart Clarke, Romberg and hosts of other pioneers? Not to surgery surely. For though men like Horsley and Cushing have advanced neurology enormously in certain directions, their work has been and must be too circumscribed to advance neurology as a whole. Neither can we hope, in America at least, for help from internists. Out internists seem to shun these important problems, and often enough boast of their ignorance of them. The American Neurological Association has numbered not more than half a dozen internists in the

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past ten years. Dr. Charles L. Dana is the one man familiar to us who, in charge of a large general hospital service, has cut his name deep into neurologic history.

NEUROLOGY IN EUROPE AND IN AMERICA

In Europe the position of the neurologist is quite different from his position with us. There neurology is looked on as an integral department of internal medicine; and those who aim to gain entrance into the ranks have had not only prolonged training in internal medicine, but they evidence their continued interest by working at it in the hospitals, and teaching it in the universities. The medical clinics of Germany have been presided over by men whose greatest fame has come from their neurological contributions. Friedreich and Erb, of Heidelberg; Nothnagel and Strümpell, of Vienna; Schultze, of Bonn; von Leyden, of Berlin; Lichtheim, of Königsberg, leaders in neurology, all were, or are, directors of the medical clinics in their respective cities. And what is true of Germany is also true in a large measure for France, Great Britain and other European countries. Witness, for instance, the contributions to internal medicine and pathology of Trousseau, Charcot, Marie, Byrom Bramwell, and of Gowers, to name only a few; or if it is necessary to enumerate other writers, the contributions that have been made by Hasse, Kussmaul, Leube, Sahli, Rumpf, Brissaud, Grasset, Head, Wyllie, and other teachers of internal medicine. Not only do their systematic treatises of medicine reveal their mastery of the intricacies of neurology, but their individual discoveries must be mentioned in headlines when the history of neurology is written.

But what is to become of neuropathology in America, where neurology and internal medicine are distinct, and where practically all the laboratories and wards are controlled by men who do not investigate nervous cases and are closed to those willing and capable to work in this field?

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The answer is not far to seek. Neurology must fall behind and cannot take its place in the coming American revival. The writing is already on the wall. We can claim no great achievement in the past ten years. American neurologists do not obtain the intimate experience with acute nervous diseases which belongs by right to the house physician in the Queen's Square Hospital in London, and we cannot hope, under present conditions, to produce men of accurate knowledge such as have made many a small German town famous.

American neurology is not only at a standstill, but its sphere is constantly being curtailed. The American Neurological Association this year refused to take part in the discussion on serum therapy at the Congress of American Physicians, on the ground that "it was not germane to neurology." Why not be frank about it and admit that neurologists have never had the opportunity of judging the effects of serum therapy on the nervous system? Instead of neurology having its share in the progress of the sciences concerned with bacteriology and with nutrition, the diseases it once claimed are one by one being taken from it. Exopthalmic goiter, a nervous disease if there ever was one, is more and more rare in neurologic clinics. As the neurologist has neither beds nor laboratories, the psychoses and neuroses with gastric symptoms have entirely passed from his hands and have lost their names. Under the mask of gastroptosis, mucous enteritis, and achylia, the patients are subjected for years at a time to the pernicious suggestions connected with local treatment.

A WIDER HORIZON IN NEUROLOGY

It is partly our own fault. During the formative period of our specialty a few years ago, the American neurologist prided himself on being a neuro-anatomist *par excellence*. To make cerebral localizations, to designate diseased tracts, to distinguish between organic and functional diseases constituted his chief occupation. The

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anatomic period has reached its zenith. To-day there is a wider horizon.

Nervous disorders and nervous phenomena are so directly dependent on and so intimately associated with derangement of metabolism, diseases of the viscera and ductless glands, to syphilis and other infections, that it is foolish to attempt to make an approximation of the pathogenesis of these symptoms unless the physician is competent to investigate and detect such pathologic states or intelligently to criticize the reports of trained men working with him. And then that great wilderness of pathology, the psychoneuroses—what opportunity is furnished us to study them intelligently and thoroughly in this country? A hurried interview in the out-patient department, where neither the environment nor the facilities favor the slightest revelation of the soul, then the patients disappear into the maelstrom of Eddyism, quackery, and the commercial sanatorium.

We are not urging a wider field for the neurologist than that of neuro-anatomist without taking into account human limitations. We recognize the time-consuming requirements of physiology, of anatomy and of special clinical investigations of the modern neurologist. We do not expect to see the neurologist, except perhaps when working at some special problem, an expert in fevers or in abdominal tumors. But we know that we are right in protesting against the narrow specialism into which he has been crowded. Unless the physician who professes to interpret nervous disorders is skilled by daily practice in making physical examinations, unless he is permitted to become familiar with the personalities of his patient through daily contact, he cannot attain that which he should attain, namely, the highest type of physician. The only way he can hope to develop such skill is in the wards of a hospital. In these wards he shall have the same facilities for investigating disease as his colleague whose inclination leads him to concentrate his clinical acumen on disorders of less noble vis-

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cera than the brain and spinal cord. There he shall have the opportunity of calling to his aid pathologist, cytologist and chemist, who shall work under his direction, and he shall avail himself of the opportunities which such service gives to study the personality or soul of his patient, without a knowledge of which therapy will always remain a barren field.

NEUROLOGIC HOSPITALS DEMANDED

Until general hospitals have neurologic wards with independent staffs, or until neurologic hospitals shall be established in every metropolitan city, we cannot hope that neurology will take the place in medical science that it deserves. We appeal to the profession to establish such services.

In New York we have organized and put into operation such a hospital, and with the hope that a brief description of its scope and organization will stimulate and encourage others, we venture to describe it briefly.

ORGANIZATION OF THE NEUROLOGICAL INSTITUTE

We arbitrarily chose three men who were responsible for the medical organization, development and direction. We readily secured a board of directors made up of men well known in business and in philanthropy—New York's first citizens. We were fortunate in finding a building adequate for our purposes, which had been constructed as a training-school for nurses and used later as a private hospital. The sum of twenty-five thousand dollars sufficed to make the necessary changes and to install the equipment for hydrotherapy, electrotherapy, Zander apparatus, electric superheated air apparatus, occupation rooms, roof-garden, clinical and chemical laboratory and autopsy room. Complete operating, sterilizing and anesthetizing rooms were already in existence. The building was subdivided into three parts—wards—with accommodations for forty patients; semi-wards, with accommodations for twenty patients, and twenty private rooms. The first floor of the building

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is devoted to the out-patient department and the therapeutic department. Here patients applying for relief are examined by the attending physicians and by salaried assistants working under them, and those whose symptoms require further study and interpretation are sent into the hospital, while those whose disorders can be satisfactorily treated in the various therapeutic departments are referred thither. Each of these therapeutic departments is under the immediate direction of physicians, and the treatment is administered by salaried assistants. In addition to diseases of the mind and nervous system, metabolic and nutritional disorders such as gout, rheumatism, arthritis, diabetes, etc., are received and treated. Other diseases are referred to the services elsewhere of the medical staff and the consultants.

The second and third floor of the hospital are devoted to the wards and semi-wards. The price of the beds in these wards, inclusive of everything, is $15 a week. Any member of the medical board may remit any part, or all, of this amount in individual cases, and about one-half the patients pay nothing. In the semiprivate wards, patients pay from $15 to $25 a week. The private rooms are reserved for patients of the physicians connected with the hospital. The revenue obtained from these rooms materially helps in meeting the expenses of maintenance. The sixth floor is devoted to the chemical and pathologic laboratory, the operating-room, the clinical and psychologic laboratory, and to quarters for the house staff. The laboratory is in charge of an expert chemist and microscopist who is paid a salary of $2,500 a year. The house staff consists of six physicians, three interns and three externs, all of whom are salaried. In addition to this staff, we have the assistance of special consultants in neurology, internal medicine and the various specialties, and an attending surgeon and an opthalmologist, both well known for their achievements in neurology.

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At the end of the first six months of the Neurological Institute's existence nearly two thousand new patients had been examined in the out-patient department and upward of three hundred patients had been admitted into the hospital. It has been no uncommon thing to give from sixty to seventy hydriatic treatments in a day; and four massage operators, two electro-therapists and two teachers of exercise and educational movements are constantly employed. The present building, which, it was at first thought, would long be adequate to meet the demands put on it, has already been shown to be entirely inadequate. The best evidence that such a hospital was needed, and that it meets a want that was urgent, is furnished by the reception it has received.

We propose soon to establish a country branch, conducted on the sanatorium principle, to which patients can be transferred from the hospital as soon as their diseases have been adequately interpreted. In the country only is it possible to carry out all of the disciplinary measures that make for the restoration of the diseased personality, the disordered functions, and the perverted metabolism, which these patients always have.

The establishment of the hospital on a working basis has necessitated attention to so many practical details that as yet we have been unable to plan the scope and equipment of the experimental and pathologic departments. These will by no means be forgotten, as we have from the outset regarded them as the important features of the institute.

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THE STATE CARE OF THE INEBRIATE

IRWIN H. NEFF, M.D.
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Although drunkenness is generally considered a misdemeanor, its association with criminality and criminal procedure has given the public the impression that the drunkard, if not a criminal in a legal sense, is so closely allied to these delinquents that the measures applicable for the control of the criminal can be consistently used in the management of the inebriate. The impracticability of such methods is recognized by those familiar with the varied types of inebriety.

Credit must be given to the judiciary and particularly to the various probation commissions for the heroic efforts which they have made to formulate some satisfactory method for dealing with the drunkard. They have recognized that the drunkard has an individuality, but owing to the diversities of the types and lack of facilities for studying these cases they frankly acknowledge their helplessness and are ready and willing to cooperate with the medical profession in any reasonable way. Massachusetts with its 90,000 arrests for drunkenness in 1909 is certainly a strong appeal for the inauguration of some plan which will individualize, differentiate and care for the habitual or pathologic inebriate.

INEBRIETY PATHOLOGIC—CLASSES

The term pathologic as applied to inebriety is expressive and eminently practical. For clinical purposes pathologic inebriates can be conveniently divided into three classes. The first group comprises men or women

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originally of normal health but who, through overwork, emotional stress and business reverses, have lowered their power of resistance. With frequent indulgence in stimulants the patient becomes powerless to discontinue his habit. This is considered to be the most curable class of pathologic inebriates. A second group considers the periodical drunkard or dipsomaniac, people ordinarily temperate, or even abstinent, who at certain periods yield to an obsession of stimulation. The third group comprises the defectives and degenerates among drunkards. Alcoholism of the patient or of his parents may in some of these cases have brought on directly or indirectly the impaired mental and physical condition, but it is equally true in other cases that imbecility, insanity and other forms of degeneracy have preceded and have been responsible for the excessive use of alcohol. The physicians in charge of the Massachusetts correctional institutions are inclined to assert that the large majority of habitual drunkards in their care, are men of less than normal mentality.

SUMMARY OF EIGHT HUNDRED CASES

Institutional statistics have proved that a predisposing cause can be almost invariably found in the pathologic inebriate. The clinical study of approximately eight hundred cases of inebriety at the Foxborough State Hospital during the past two years is my justification for the following summary. These conclusions may be considered as an attempt to establish a clinical entity for inebriety.

1. Inebriety is an expression of nervous weakness or nervous instability; used in its simplest sense it could be called a psycho-neurosis, many cases showing symptoms which are found in neuresthenic states and allied conditions. Addiction to alcohol is a symptom of an unstable nervous system and the contrary view expressed by the laity is not justified by clinical observation or experience.

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2. The exciting causes of inebriety are of a physical and psychic origin. Given a neurotic subject crises may be precipitated by any marked departure from the ordinary routine (psychic) or by any disturbance of organic nature (physical).

3. Inebriety is prone to develop as a frank case at the critical epochs of life, namely, during pubescence, adolescence or involution. Developing during involution it is generally the effort of an individual to maintain his productive power by recourse to artificial stimulation. The frequent inception of inebriety at these periods suggests an analogy to the psychoneuroses.

4. Inebriety being an expression of neuropathy or psychopathy, may be preceded or accompanied by a multiform nervous syndrome; thus each case is essentially different.

5. The heredity element in inebriety is considerable and is undoubtedly a powerful predisposing cause. A history of decided intemperance in the parents existed in over 40 per cent. of our cases, while 15 per cent. gave a history of defective ancestory, insanity, neuropathy, drug addiction or tuberculosis being present on the maternal or paternal side.

6. Approximately 5 per cent. of our cases showed preexistent mental symptoms which could be differentiated. Some of these were distinct cases of psychasthenia; others were of the milder forms of manic depressive insanity.

We feel warranted in corroborating the statement made by others that an inherited neurotic or psychic tendency is present in a considerable number of inebriates. Many of these cases admit vagaries of conduct and peculiarities during pubescence and adolescence, showing clearly neurotic or psychopathic conditions which have antedated the alcoholic manifestation. We venture to say that our future study will support these conclusions.

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INDIVIDUALIZATION IN TREATMENT

The necessity for individualization and differentiation in cases of drunkenness is made more evident if we inaugurate any method of treatment. It seems unnecessary to say that such differentiation should be controlled and directed by the medical profession. The successful treatment of inebriety depends on the accurate discrimination and individual consideration of cases. These conditions can be more satisfactorily brought about in institutions under state supervision and control.

The defects in existing methods of care of the drunkard, which methods are in great part criminal, are recognized in Massachusetts. Her experience may be justly considered to be similar to that of other states. While release, suspended sentence, probation and payment of fine while on probation, is an ideal method in some cases, it can be said without fear of contradiction that imprisonment for drunkenness and non-payment of fine are futile as a method of reform and useless as a means of protecting the public. The large number of recidivists in alcoholism bears testimony to this assertion.

The habitual drunkard is omnipresent and resistive to the ordinary methods of treatment of reformatories. R. W. Brenthwaite, in the report of the inspector, under the inebriate's acts, 1910, expresses this clearly when he says that owing to educational measures and popular agitation, there has been a considerable and progressive improvement in the general drinking habit of the nation. The drunkenness that has been rendered less common in England is the occasional vicious excess of the man who can remain sober if he will. The large majority of the population, owing to educational measures, are now moderate drinkers who rarely get drunk, a case which is apt to engender a feeling of satisfaction until we look below the surface and observe the army of habitual drunkards which may be properly termed pathologic inebriates. This also emphasizes what I be-

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lieve every medical man can verify, that it is by no means clear that any real advance has been made toward the suppression of habitual drunkenness.

The increasing prevalency of drunkenness is responsible for the concerted movement for the care and treatment of inebriates in England some thirty years ago. They have devoted time and money to the elucidation of this problem. At the present time there is under consideration progressive legislation which will increase the usefulness of their methods. Australia, Germany, Switzerland, Hungary and Denmark have laws which provide for the care and treatment of the inebriate. Recent correspondence with these countries shows that they are making steady progress and that the authorities are keenly alive to the fact that state and municipal care of the inebriate is not only a reform measure but is of decided economic value.

The need of state care of the inebriate was almost simultaneously recognized in the United States. Massachusetts in 1893 established a hospital exclusively for the treatment of the inebriate. Iowa and Minnesota have established similar institutions and in many states provision has been made for the compulsory commitment of inebriates to state institutions. During the past year many states have appointed committees for the investigation of drunkenness and with power to advise on conditions and remedies. Maryland, Michigan, Connecticut, Ohio, Indiana, New Jersey and New York are actively concerned in such investigation. Pennsylvania is engaged in this reform and undoubtedly will eventually succeed in carrying out the purposes of the bill so actively championed by her medical profession. New York City has recently passed a bill which provides for the care and treatment of the inebriate; the proposed scheme is perhaps the most elaborate which has thus far been advanced and its inauguration will be looked for with interest. New Jersey, although meet-

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ing with reverses in her attempt to secure state care, is hopeful of success and has been conducting a good publicity campaign.

CONSIDERATIONS OF CURE

When we consider the permanency of benefit from institutional treatment we must remember that inebriety is a chronic disease, and bearing in mind our conception of inebriety, namely, that it is a state of nervous weakness plus a habit in which the patient has a graded responsibility, we can readily appreciate that the word "cure" must be used relatively and that the condition which we call inebriety is subject to the same inconsistencies which we find in many cases of functional nervous disorder. When considering the benefit which may result from institutional treatment it is necessary to consider the type of inebriety, the time of abstinence, and to compare the man's present mode of living with his daily habits prior to his admission to the hospital. This plan has been carried out when studying those discharged from the Foxborough State Hospital. I am not prepared to give statistics on"cure" but I can conscientiously say that a large percentage of cases have been permanently benefited by treatment.

STATE HOSPITAL TREATMENT

A state hospital should care exclusively for the hopeful cases and these should be carefully differentiated from cases not likely to be benefited by hospital treatment. The following conditions regarding state hospital treatment of inebriety are based on our experience during the past two years.

1. Individual consideration of each case is the only rational and effective mode of treatment.

2. Abstention from alcohol is not sufficient for cure; it is required that the patient cooperate in normal measures instituted for his betterment and the ultimate success of hospital treatment depends on this sustained interest.

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Medical after-care is necessary to obtain the maximum degree of success. This medical after-care to be of any benefit must be practiced with the consent and cooperation of the patient.

A state hospital for the treatment of inebriety should be considered an educational center; it should have adequate equipment for treatment of such cases, and should have facilities for segregation and individual treatment of the diverse types.

The word "hospital" when used in connection with inebriety is considered to be synonymous with "cure" or "improvement." Used in this sense it can be emphatically said that, with modern methods, hospital treatment of inebriety by the state is of economic value.

IMPROVEMENTS IN THE LAWS

The experience of Massachusetts during the past five years has revealed failures in the existing conditions which has served as a basis for pending legislation.

The trustees of the Foxborough State Hospital have presented a report to the general court, now sitting, which was submitted in accordance with legislative resolve approved during 1909. This legislative document has been presented with accompanying recommendations and a bill asking for an appropriation for the purchase of a site for the establishment of a new institution has been introduced. The report involved the careful study of the treatment of inebriety in this country and abroad; it considers in detail drunkenness in Massachusetts, its conditions and remedies. The following is a summary of the recommendations:

The Board of Trustees of the Foxborough State Hospital in their forthcoming special report, prepared in accordance with Chapter 111 of the Acts and Resolves of 1909, offer a statistical study of drunkenness in Massachusetts with reference to the cost of inebriety to the commonwealth. In place of the present system of fine or sentence to prison for the frequent offender, is substituted a plan whereby the non-criminal drunkard may be completely segregated from the

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other offenders of our penal system and the curable cases among them given every opportunity for cure. The plan further includes provision for the segregation and cure of female drunkards who have hitherto been subjected to custodial treatment only.

To this end the trustees of the Foxborough State Hospital have recommended to the Legislature of the Commonwealth the following:

1. The establishment of a new state hospital colony for inebriates to receive:

A—patients who come voluntarily (Acts of 1906, Chap. 316).

B—cases committed under Sec. 59 of R. L., Chap. 87.

C—young habitual drunkards placed on probation by the court on condition that they spend their probation period at the hospital.

D—suitable cases transferred on parole from the detention colony.

2. The establishment of a detention colony for incurable non-criminal inebriates, to be situated in a town neighboring to the hospital. To receive:

A—Incurable cases committed as under R. L., Chap. 87,

B—confirmed drunkards on indeterminate sentence.

3. The establishment of a hospital colony for non-criminal female inebriates to receive:

A—voluntary cases,

B—cases committed under R. L., Chap. 87,

C—cases sentenced from the courts.

A part of the institution might be devoted to the incurables of Class C until the state had sufficient experience with provision for female inebriates to ascertain the need of a separate colony.

These three institutions for purposes of facilitation of transfer and for uniform management should be under the same board of trustees and the same medical direction.

This system as above outlined provides for all types of drunkards beyond the release and probation stages, except for the drunkard with criminal taint, who, together with refractory cases of the detention colony, should be sent to the state farm.

The present Foxborough Hospital is inadequate for this extended purpose, because of general lack of equipment and facilities. Some of the most obvious lacks in view of constructive treatment are:

1. Insufficient land for agricultural development.
2. Insufficient plant for industrial training, etc.
3. No opportunity for segregation of diverse cases.

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The advantages of proposed system are:

1. A large per cent. of the 20,777 persons sentenced in Massachusetts in 1909 for drunkenness are curable. By this system such cases could be ascertained and the patients cured.

2. The utter waste of state resources involved in the sending of 9,958 cases in 1908 to prison for a month or less on account of non-payment of fine could be obviated. Some of these men could be cured permanently. The others could be kept apart from criminals and put to work that would largely pay the cost of their maintenance—a cost which today, including expense several times per year per individual drunkard for arrest, trial, transportation and detention, is very serious.

3. The woman drunkard, who is now forced into houses of correction or state reformatories with thieves and prostitutes, could have treatment for cure whenever curable, and in any case could be kept from the street, on the one hand, and from association with criminals on the other.

The adoption of this plan will eventually lessen materially the cost to the state for the care of the inebriate. The experience of Massachusetts in caring for drunkenness is undoubtedly paralleled in the majority of states. The direct cost to the Commonwealth of Massachusetts in 1908 for maintenance in prison of such inebriates as were sentenced on the charge of drunkenness was over $500,000. When we include the cost of arrest and trial, and particularly the cost to the public and private charities for maintenance of families, it is safe to assume that these additional expenditures place the direct annual cost of drunkenness to the city and state at a point far beyond $1,000,000. This vast sum of money expended for arrests, arraignment and imprisonment is certainly an appeal for better discrimination and a more rational mode of procedure in taking care of these people.

ABSTRACT OF DISCUSSION

Dr. Alfred Saenger, Hamburg: I am glad to hear that in New York there is now a neurologic institute. Erb was the man who said that it was necessary to found such neurologic institutions. In Germany we do not have them, but in Hamburg we have neurologic wards in the two large general hospitals. In the one hospital at Eppendorf there is my colleague, Dr. Nonne, and in the other, the Hospital of St. George, I have

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a number of beds, and a so-called polyclinic for outside patients.

Dr. Collins states that the internists have the lead in neurology in Germany. That was so formerly, but later on we have had neurologic specialists such as Oppenheim and others, who are not internists; and it seems to be true that neurology is really a department by itself; and Erb said, at the last association meeting held at Vienna, that the time would soon come when there would be founded in Germany chairs of neurology, where formerly chairs were instituted for the internal physician exclusively. Three years ago Oppenheim, Bruns, v. Frankl-Hochwart, Edinger, v. Monakow, the late Moebius and myself established the Association of German Neurologists, and we had the pleasure at our meeting last October at Vienna of seeing several American neurologists.

Dr. Charles L. Dana, New York: The work accomplished in the establishment of a neurologic institute has been most praiseworthy and satisfactory and the importance of such an institution cannot be exaggerated. We have just had enacted a law which enables New York City to undertake a certain experiment in the care of inebriates, and on the statements of Dr. Neff and the success of such work as he has done depends largely the question of whether we are going to succeed or not. The New York law is not mandatory, and we have to go before the board of estimate and apportionment and ask them to give us money to carry it out; and if Dr. Neff's results and the experience of others in this work show that it is of economic advantage we can get the money. It is just a question of economics. In New York we have a bad history in so far as practical care of the inebriate by state institutions is concerned. This failure of the state institutions, several years ago, however, was due to politics and defective laws and administration; it is a *sine qua non* in the care of the inebriates that the institution be taken away from politics and that it be under the control of an intelligent board of managers. If any city or state cannot begin that way it might as well not begin at all. In New York we are trying to inaugurate the farm colony plan as applied to the inebriate, and instead of having the drunken man punished as a criminal we propose for the first and second offenses to let him go on probation, and then on the third offense to send him to the hospital, and then to a farm, where he is committed on a somewhat indeterminate sentence.

The whole question is one which should appeal to neurologists and interest them.

Dr. T. D. Crothers, Hartford, Conn.: It is a source of great satisfaction to have this subject come into prominence again. When the asylum at Binghamton, N. Y., the first ever organized in the world for the treatment of inebriates, opened in 1863, it excited intense opposition. Medical men and laymen called it a quack effort, denounced the theory of the disease of

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inebriety and its defenders as unworthy of notice. Its early presidents, the late Dr. Valentine Mott and the late Dr. Willard Potter, were condemned for having anything to do with the work. Boss Tweed was a trustee at one time, and the institution went into the hands of politicians, who after sixteen years of troubled experience turned it over to an insane asylum. The questions which Dr. Neff has brought out were discussed at that time, with equal clearness, but they were too far ahead of public opinion to attract any attention. My early experience began as an assistant physician in that institution in 1875. I assisted in making the first statistical study of the history of 1,100 patients who had been under treatment at the institution nine years before. The returns showed that 60 per cent. had continued sober and temperate up to that time. These statistics were condemned as fictions, and yet after the long lapse of time they have been confirmed, and were really the first scientific studies ever made. The curability of such patients was established, but there are people to-day who doubt it and interpose obstacles and criticism. Massachusetts is facing the same problem that came up in New York fifty years ago. I have for over a quarter of a century discussed these very questions in this Section in many papers. Now a new generation is coming on and their practical recognition is assured. The best statistics show that from 20 to 40 per cent. of 5,000 new cases of insanity registered last year in New York City are due directly and indirectly to alcohol, and this cause of all others must come into the field of medical study and critical examination.

It will be of interest that the government has published the transactions of our Society for the Study of Alcohol and other Drug Narcotics, as Senate Document 48, a book of 135 pages, containing thirty-two articles on the economic, scientific, hygienic and medical sides of the great alcoholic problem, which is the best collection of modern literature published on this subject. This is the first time the government has ever published the transactions of any medical society and indicates the profound interest in the subject. The document is sent free to all who ask their senators at Washington for it.

Dr. F. P. Norbury, Kankakee, Ill.: The organization of the Psychopathic Service of Illinois follows the line suggested by Dr. Collins. The institutions of Illinois, including the Psychopathic Institute, located on the grounds of the Kankakee State Hospital, are on a civil service basis. The organization of the service is educational; the young men are encouraged to enter the service as interns, then after a year's experience, to take the promotional examination for the grade of assistant physician; then after a service of a few years, they are eligible for promotional examination to the grade of physician; then later to the grade of assistant superintendent.

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The Psychopathic Institute has a staff as follows: Director, psychologist, clinical pathologist and chemist. The work of instruction embraces the methods of clinical psychiatry and neurology, approached not only from the standpoint of psychology, but of internal medicine as well (a training in diagnostic methods of internal medicine), and consideration of advanced methods of treatment, including bedside studies suggested by Dr. Collins. To insure recognition of the educational values of such a training and its appreciation by the people whom we serve, we must have the cooperation of the medical profession. We of the west are hopeful of the continued advancement of clinical psychiatry, inaugurated by Michigan, encouraged by Indiana, Illinois has enlisted for the full values which may accrue to our profession and those whom we serve, in this education work.

Here in Missouri, under the leadership of Bliss, a similar campaign has been inaugurated, which we all hope will come to fruition, thus adding to the promising future of clinical psychiatry in the west, based wholly on a strictly professional and educational basis.

Dr. Frank R. Fry, St. Louis: We should show all the enthusiasm over this matter of legislation concerning inebriates that it is practically entitled to, but not more. Dr. Dana sounded the right note when he said that we should watch closely the practical workings of legislative attempts, and that future attempts should be based on statistics, if they will help us, or on other considerations of what has been attempted in this line.

We ought to be careful of our statements, action and recommendations, because they will be accepted as somewhat authoritative. For instance, one of the speakers on the floor just now said that certain statistics showed that a certain proportion of insanity was due to alcohol—20, 30 or 40 per cent. of cases. Now, I do not believe that there is any statistical way of proving that thing; and therefore, we ought not to say so.

As Dr. Crothers and Dr. Dana have said, attempts at legislation in this direction began many years ago, and they were failures; it is very demoralizing to attempt to do something of this kind and fail.

Dr. Hugh T.. Patrick, Chicago: I am sure we are all enormously indebted to Dr. Collins and his colleagues for making this dream of a neurologic institute come true, because a dream which has come true is an enormous lever with which to pry loose the necessary funds to make more dreams come true. A demonstration beats a theory all to pieces. And I wish to say just a word of encouragement to this pessimistic Dr. Collins, for there are even now schools in which the ward walks and bedside section work are not unknown. We have them out where we live. I am bound to say that our material is not always adequate, but we have made a start.

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We would have more neurologic services, pure and simple, if the neurologists were a little more aggressive. The truth is, we are too considerate of the feelings of the internists. I think that every neurologist ought to be willing to say right out in meeting that he can diagnosticate and treat a case of apoplexy or tabes or any other nervous disease better than an internist can; that he can better demonstrate it to a class; that he can talk about it better; that he can instruct the student more effectively than any internist on the faculty. If the neurologist cannot do this he ought not to be on the faculty at all. In Cook County Hospital, our largest city hospital, we now have a real neurologic service with neurologists on the staff who have their own patients and the entire control of them, also the control of the post-mortem material and can use it fully and without restrictions. They have their allotted interns as well. We have not yet our patients segregated in separate wards, as they should be; but after all, segregation is not an unmixed benefit. When the cases are mixed in the wards, the internists are apt to see them and the neurologist is apt now and then to see a case of the internist. If we neurologists did better and more fundamental work, according to our opportunities, then we would soon have more. In my experience the internists have always shown themselves not only ready for but anxious to have the assistance of any neurologists who were willing to sit down and spend the necessary time with them; and there's the rub. One of the great advantages that Dr. Collins has in his new institute is the paid assistant. Our work is such a time-devourer that unless the best men, the thinkers, the men with ideas, can have assistants who shall not be asked to spend thousands of hours for nothing, we cannot make the sort of advancement which we should make and we shall be a long time in arriving at such a wonderful organization as Dr. Saenger and Dr. Nonne have in Hamburg.

Dr. Haldor Sneve, St. Paul: I have to take issue with Dr. Neff and Dr. Crothers about inebriety being a disease. I think that as long as we maintain that inebriety is a disease we are keeping back the hands of the clock that marks progress in the treatment of this condition.

I would make three classes of inebriates, and I would put the vast majority, say three-fifths of all inebriates, in the class of inebriates from social habit. Inebriety is a habit seldom acquired in middle or old age, but most frequently in youth, from bad associations. To see where inebriates are trained, all one has to do is to go into the slums and saloons and clubs and see there the young men.

The second class of inebriety is the neurotic type. I do not believe that the neurasthenic or neurotic inebriate, or the neurotic of any kind is a born neurotic; I think he has acquired the neuroses. I do not believe that we are born

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with neurasthenia, hysteria, or any of these things; we acquire every one of them. We have an immense number of neurotics in our society; every family has neurotics, and no statistics on heredity will help us very much on this point. But the neurotic will become a morphino-maniac, a cocain fiend, or "slop-over" in some way.

The third class is that of periodic drinker. If there is anything in the mental condition or disposition which makes an inebriate it must be in the dipsomaniac so-called, who goes on sprees at intervals, and the rest of the time is a good, upright citizen. If heredity played a preponderating rôle in inebriety, most people would be inebriates, women as well as men, because our grandfathers and great-grandfathers, and great-great-grandfathers before them were all drunkards practically—at any rate nearly all heavy drinkers. The statistics that have been piled up, especially in France, about the influence of alcohol on the production of the inebriate, and the criminal, and pauper, and other departures from the normal, I think are absolutely misleading. Why, in the rural districts in Europe, at the weddings and other feasts everybody got drunk, and especially the groom, and the first child might frequently be conceived while its parents were in a drunken state. We believed for years and years that if the parents were drunk at the time of conception the child would be born a drunkard, or a hysterical lunatic or defective of some kind. I do not believe it. Does anybody contend that a taste for tobacco is hereditary?

In Minnesota we are building an Inebriate Farm, believing that drunkenness is a bad habit. Somebody wished to know how to get money from the state to build an institution of this kind. We drew up a bill and went to the Board of Control of Minnesota with it; and we inserted a provision that the hospital was to be built and maintained by a 2 per cent. tax on all saloon licenses. A legislative appropriation will be delayed for years and years; all one has to do is to propose to take a tax from the liquor traffic which produces these inebriates.

Dr. Herman H. Hoppe, Cincinnati: Every neurologist should go home with the firm determination to try to establish in his native city some neurologic service in the city hospitals. We have had in Cincinnati neurologic service for ten years in city hospitals, a separate ward for the males, and part of a ward for the female neurologic patients. I do not believe that the standing of neurologists will ever be on a firm basis until they maintain that they are, as Dr. Patrick says, more capable of making neurologic diagnoses and taking care of neurologic cases than the internist who calls them in for consultation. When Dr. Collins was in Cincinnati a few days ago, I showed him two cases of obscure family paralysis which no neurologist could diagnose by going into a hospital

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on neurologic service and giving fifteen minutes to this case. They would require continued study for hours and perhaps days and weeks before a definite opinion could be had which would be worth anything. Being merely a consultant will not give one the standing in the hospital or perhaps the opportunity or the desire to go back and study these cases as they should be studied.

Our system is as follows: If a man on another service wishes a neurologic consultation, he makes a written application for it, and then the case is either transferred to the neurologic service, or it remains a joint case until disposed of either by diagnosis or by some operative treatment, if it should happen to be an operative case; and I believe that this is the only satisfactory method by means of which neurology can be practiced in cities where there are general hospitals, and the only satisfactory practice which will give the neurologic specialist the standing to which his past and present work entitles him.

Dr. Joseph Collins, New York: My contentions briefly are that neurology is an integral part of internal medicine, and that the only way to be a neurologist is to have it a specialty, founded on internal medicine. It would be impossible for me to practice neurology without being an internist; and therefore, I have had for fifteen years, and hope to have until I retire, a service in a large general medical hospital. I appreciate that in certain parts of the country that does not exist, and that a neurologist in certain parts of the country is a man who treats neurasthenia only, and in other parts is a man who treats neurasthenia and mental disorders.

I am familiar with city hospitals, such as the Cook County Hospital and the Cincinnati General Hospital, because I have been for eighteen years an attending physician to the New York City Hospital, which is very similar. Such hospitals do not get the class of cases in neurology with which we are taken up in private practice. It is no longer a large part of our work to diagnosticate tabes or syringomyelia; our work is centered chiefly on the consideration of and the effort to effect cures in the so-called borderland cases of neuropsychosis, and cases in which it cannot be determined whether there is an organic disease or a metabolic condition. They come only to such hospitals as the Presbyterian Hospital and St. Luke's in New York, and other hospitals similar to those in your own cities that receive acute cases. In those hospitals in New York, Philadelphia and Boston, the cities with which I am familiar, there is no neurologist who has any welcome or right save as a consultant. There is no neurologist connected with any such institution who has the privilege of going into these hospitals except on special invitation, and there he is expected to deliver an *ex cathedra* opinion, not based on an adequate history, but merely on a short examination. In reality he is not competent to deliver such an opinion within a month or within two or three months;

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and he should be permitted to have the opportunity to study the case in order that discredit should not come to his diagnostic acumen.

I am not pleading that neurology be given up as a specialty. I am maintaining that its progress has ceased, because the internist has not looked on it as a part of medicine with which he should make himself familiar; and on the other hand, he has attempted to overlook neurologists by keeping them out of hospitals to which they are entitled to go. The only way left is to insist on the establishment and development of hospitals for nervous diseases, and on having neurologic wards in general hospitals for the treatment of acute diseases.

Dr. L. Vernon Briggs, Boston: In regard to the physiologic work in the hospitals in Boston I have felt for a long time that the work of neurologists and alienists should be separate. I have been in charge of a mental clinic twelve years in Boston alongside of a nervous clinic; we are entirely separate; and the nervous and mental clinics in the London hospital are separated. They treat 1,000 patients a day. It is not so in most of the hospitals; the nervous and mental cases are treated together or by the same physicians.

In regard to what has been said of the Boston hospitals by Dr. Collins I take exception, in that Dr. James J. Putnam has this last year among his friends secured a fund for the establishment of certain beds for neurologic patients in the Massachusetts General Hospital. These were placed entirely under his control. Patients to be sent by the neurologic department to those beds can remain there as long as the physicians want them to. Following the Massachusetts General Hospital the neurologists of the Boston City Hospital, through Dr. Prince mainly, have persuaded the trustees to set aside twenty beds for their clinic. These are entirely devoted to nervous and mildly insane patients.

Dr. Irwin H. Neff, Foxborough, Mass.: I think that the discussion on inebriety has proved that there is a lack of uniformity of opinion in the medical profession on this subject. But it makes little difference whether we consider it a disease in a special or general sense, or whether we consider it purely a habit. My opinion is that it is a habit engrafted on a nervous condition, but, be that as it may, I cannot help but emphasize that the need of state care for the inebriate is as great as that of caring for the insane. It is only within the past twenty years that the medical profession has taken up the question of modern psychiatry; any advance made in psychiatry during the twenty years has been wholly due to the interest taken in it by the medical profession, and I want to appeal to each member of the Section to work for state care of the inebriate. I think this should be considered by each member of the Section from a clinical, sentimental and economic point of view.

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THE TOPOGRAPHIC DIAGNOSIS OF SUBTENTORIAL BRAIN TUMORS

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The great advance in brain surgery in recent years has been that concerning the subtentorial region. Many new signs and symptoms have been put forward as throwing new light on the topographic diagnosis of tumors beneath the tentorium cerebelli, and I believe that it will not be a waste of time to present illustrative cases and to subject the syndromes of tumors in various localities in or about the cerebellum and brain stem to a critical analysis.

TOPOGRAPHIC DIAGNOSIS

The question as to whether or not we shall operate for tumors in this region, and if we operate, whether the operation shall be radical or palliative, can be decided only by an accurate topographic diagnosis. The subject of differential topographic diagnosis is therefore of prime importance. Surgical intervention is dangerous, or not, in the cerebellar region in direct ratio to the amount of exploration, handling and bruising to which the cerebellum is subjected. Moreover, if we know, from the location of the growth, that the chief disturbance produced is by an internal hydrocephalus, as in Case 1, the nature of the surgical interference will be different from that which is indicated if the growth itself, by local pressure, is threatening the life of the patient. More accurate topographic diagnosis will not

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only enable us to be more precise in advocating or refusing to advise surgical intervention, but it will enable us to say whether the suboccipital or subtemporal region is to be the seat of the operation, and, if suboccipital, which side.

Have we signs sufficiently positive to enable us to say that a growth is in the brain-stem, in the cerebello-pontine angle or intracerebellar, and if the latter, on which side it is located?

The cerebral surgical triumphs of the last decade have been achieved in relieving cysts and tumors of the cerebellum itself, and those of the cerebello-pontine angle. The diagnosis of the latter growth as we shall see is comparatively easy. It shall be the chief object of this paper to analyze the signs and symptoms which indicate a lesion of the cerebellum itself. I shall assume that such a localization can be arrived at only by exclusion, and I shall therefore briefly state the history of cases, in which there were illustrative lesions, which must be excluded in the topographic diagnosis of intracerebellar growths.

TUMORS OF CORPORA QUADRIGEMINA

The first case to which I will call your attention and show you diagrams illustrating the pathologic findings is typical of tumors of the region of the corpora quadrigemina. I will give but a brief outline, because the case itself will form the basis of a future paper.

CASE 1.—*Patient.*—E. C. H., aged seventeen, school-girl; parents absolutely well. With the exception of the diseases of childhood, she had always been robust. About one year ago patient began having headaches. Two months later she began to have pain in the eye-balls, and about the same time she began to see double. The pain was chiefly located in the frontal region and on the right side. When walking she would often stagger to the right side. The right arm and leg, although not weak, did not seem right. In September she began to lose vision, and in December became totally blind. The patient often had hiccoughs, and vomited almost daily, especially when the headaches were violent. She at times

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had sudden attacks of rigidity of all her muscles, fell to the ground, but did not lose consciousness.

Examination.—April 19, 1909. Patient's mental condition was good. There was no pupillary response to light; atrophy of both optic nerves, with choked discs; paralysis of both external recti. The external muscles of the left eye were all paralyzed except those which move the eyeballs downward. She was unable to raise the right eye upward or turn it outward, but it could be turned inward and downward. There was slight ptosis of both eyelids; no anesthesia of either cornea. There was static ataxia, and when patient walked she showed a tendency to fall to either side, but mostly to the right. There was some weakness of the left leg; otherwise the examination of the extremities was negative. There was no defect of hearing.

Diagnosis.—Subtentorial growth of uncertain location.

Course of Disease.—An operation was asked for by the father and daughter, but was deferred because of the hope of giving relief by the use of drugs. Relief was obtained by the use of acetylsalicylic acid (aspirin) and codein sulphate. The patient quickly lost her ability to walk at all, on account of extreme ataxia, showing a tendency to retropulsion and falling backward. Somnolence increased. Incontinence of urine and feces set in. The headache and vomiting suddenly disappeared, and for two months before her death did not return. On August 12th patient died, a few hours after an attack of coma.

Autopsy.—Limited to the cranial cavity. There was very little cerebrospinal fluid. The cortex was anemic and the convolutions were flattened. Both lateral ventricles were dilated, and the right one was filled with a large blood-clot, which had caused the exitus. The floor of the right lateral ventricle over the basal ganglion was covered by a soft tumorous mass, very extensive, but not very thick, and hardly infiltrating the thalamus. The main mass occupied the region of the corpora quadrigemina, completely obliterating the canal of Sylvius, and extending backward into the substance of the pons and laterally affecting the margin of the right lobe of the cerebellum. The rest of the cerebellum, both lateral and middle lobes, were uninvolved. The tumor was not more than an inch in diameter.

I am indebted to Dr. C. I. Lambert, of Ward Island, New York State laboratory, for the pathologic investigation of this case.

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We have here a typical case which illustrates the syndrome of tumors of the corpora quadrigemina: Paralysis of the external eye-muscles, involvement of both eyes, inability to turn eyeballs upward, ability to turn them down, pupillary paralysis, ptosis of both lids, blindness, incoördination of gait, some staggering static ataxia; no loss of sensation or of motor power in the extremities. The only discordant note is that deafness is often associated with lesion of the corpora quadrigemina, but in our case hearing remained normal. The other unusual feature is the extreme ataxia which came on toward the end. Patient when in bed used arms and legs freely but was unable to stand at all on account of the ataxia, and showed marked retropulsion on attempting to walk. Intention tremor, athethosis and nystagmus were absent in this case. The autopsy showed that the tumor was practically limited to the corpora-quadrigeminus region.

TUMORS OF PONS

Tumors of the pons offer quite a different syndrome from that of the tumors of the region of the corpora quadrigemina as the following case will illustrate:

CASE 2.—*Patient.*—C. W., aged 12, school-girl. Family history was negative. There was no tuberculosis in the immediate family. Patient was well up to four months ago. Onset of trouble was marked by a slight defect in the right eye; patient saw double; at times had some fever; headache occasionally at first; later, more frequent, and just previous to the time she was first seen had attacks of vertigo, and staggered; had vomited every morning. No defect of hearing.

Examination.—At the time of examination the mental condition was normal; pupils equal and responded to light; optic discs normal; there was paralysis of both internal and both external recti muscles; the eyes could be moved upward and downward; paralysis of the right facial, both upper and lower branches; right eye could not be closed. The tongue deviated to left; arms were normal in all respects; in the right leg the patellar reflex was practically abolished; left patellar reflex exaggerated; left ankle-clonus, bilateral Babinski sign; no weakness in legs; hearing normal; when eyes were closed patient walked with a staggering gait, with a

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tendency to turn toward the left. There was no static ataxia, no ataxia in recumbent position.

Course of Disease.—Two weeks later the condition remained the same, but a horizontal nystagmus had been developed, on looking to right. After four weeks there developed an ataxia of both arms, without any loss of muscular power; with blindfolded eyes patient could not walk forward at all, but invariably was forced toward the left. She staggered to left when walking, and fell to left in Romberg test. Hearing had become defective in the right ear, both to bone and air conduction. Papillæ remained normal; left eye movement upward somewhat diminished; left patellar reflex had returned and both were now exaggerated.

May 24, 1910, mental condition was good; optic discs normal; eye muscles as before; paresis of soft palate; patient choked on swallowing. Right side of face remained unchanged; no loss of sensation anywhere; no areflexia cornea. Speech was difficult; dysarthria was present; patient developed some-weakness, but a very acute ataxia of the left side, both arm and leg; adiadkokinesia of the left arm was marked; patient was unable to walk without assistance on account of the left-sided ataxia. Ankle-clonus was present on the left side; ex aggerated patellar reflexes.

This case presented the typical history of an infiltrating tumor of the pons. The damage wrought is caused by a gradual destruction of the various nuclei and structures of the pons, partly by invasion, partly by compression.

The symptoms are chiefly bilateral; viz., bilateral involvement of the eyes, bilateral ataxia of the arms, bilateral increase of the patellar reflexes, but the involvement of the seventh and eighth on the right side, paresis of the left leg with ankle-clonus on the left side and ataxia of left arm and leg later on, shows the typical condition of pontine and medullary growths, viz., paralysis alterans.

TUMORS OF CEREBELLO-PONTINE ANGLE

And now let me call attention to tumors of the cerebello-pontine angle. The typical tumor here is of very slow growth, usually a fibroma or endothe-

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lioma, growing from the trunk of the auditory nerve; but the seventh and fifth may also be the seat of origin. The typical early syndrome is that of defective hearing on one side, gradually increasing, with occasional attacks of vertigo, with loud noises and ringing in the ear; then the general symptoms of headache, vomiting vertigo, unsteady gait, plus more or less pronounced involvement of the fifth, sixth, seventh and eighth cranial nerves, on one side, with perhaps an indication of ataxia of arm or leg on the same side, and finally, signs and symptoms which point to a compression of the medulla and pons. This syndrome is so typical that in the majority of cases the localizing diagnosis is easy. Let me here introduce a typical case:

CASE 3.—*Patient.*—R. M. R., aged 49, examined July 9, 1908. Father of four children; superintendent of Normal school, state of Kentucky; patient of O. L. Smith, Lexington, Ky. He had had meningitis (?) when 9 years old; no venereal diseases; influenza yearly for five or six years; had been partially deaf in the left ear for the previous six years. Dr. Smith wrote that in March, 1907, patient had "some contraction of the field of vision, occasional sinistral hemianopia, dextral arm and leg numbness, and inability to nominate with his usual facility." Patient stated that for the previous year he had had occasional attacks of vertigo and could not see out of the temporal half of the left eye; that he could not hear well with his left ear. He staggered at times, but to no special side. Lost confidence when he attempted walking in the dark.

Examination.—Mental condition, memory and speech normal; left pupil somewhat larger than the right; both responded to light and accommodation; no choked disk; some weakness of internal rectus of right eye and external rectus of left; horizontal oscillations of eyeballs when turned to extreme left, but no typical nystagmus. The patient could not hear well in left ear, but bone conduction was normal. Sensation in the facial region was normal; reflexes of cornea normal. No loss of function in regions supplied by the other cranial nerves. Sensation, muscular power and reflexes of the extremities normal; no ataxia in upper or lower extremities.

Diagnosis.—No positive diagnosis could be made; neoplasm suspected.

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During his vacation patient noticed that his gait was slightly ataxic. In October and November, 1908, he suddenly fell on two occasions. His legs simply gave way under him; was able to arise unassisted and go on with his work.

Feb. 10, 1909, patient stated that he had some headache and vertigo in the fall of 1908; that he was unsteady on his feet and staggered occasionally as if drunk. Six weeks prior to above date he had a sudden onset of violent headaches and vomiting. This continued since; patient was confined to bed and treated for uremia.

Examination.—Patient had not been able to walk without support, and when put on his feet staggered and fell to the left. He was very weak; pulse 96; vomited constantly, and had violent headaches. Pupils reacted to light. Patient had very marked choked disks, but could see well; stated that vision in left eye became blurred occasionally; weakness of internal rectus of right eye and external rectus of left eye; horizontal wavering of eyeballs when turned to the left. (Nystagmus?). He showed a tendency to lie on left side; gait weak and staggering, and fell to the right; no typical cerebellar gait. Nasolabial fold seemed less marked on right side than on the left. No ataxia of the arm; right arm normal in muscular power; the right leg was perhaps a little weaker than the left; urinalysis negative.

Course of Disease.—For two weeks patient stopped vomiting; then vomited occasionally. Seemed much improved on treatment of iodid of potassium. No new symptoms or signs developed for six weeks.

April 5, 1909: Rapid increase, rather suddenly, of weakness in the legs; when put on feet, fell to the right and dragged the right leg. Reflexes of legs exaggerated; had suddenly developed bilateral ankle-clonus and slight patellar clonus; plantar reflex normal on both sides; no disturbance of sensation, no ataxia in the recumbent position, no weakness of arms; other signs and symptoms remained as before, except pulse, which was very weak; 120 per minute.

Diagnosis.—Cerebellar tumor, probably on the left side. Dr. Ransohoff was called in to operate. Dr. Zenner also saw the case.

Operation.—On account of the weakness of the patient, it was decided to do a typical subtemporal decompression on the right side, rather than take the increased risk of perhaps a bilateral suboccipital operation. The bulging of the dura was enormous; dura opened and pulsation in brain reestablished; patient left operating room barely alive, on account of cardiac

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Fig. 1, Case 2.— The photograph illustrates paralysis of right external rectus, right facial, and left-sided hemiataxia.

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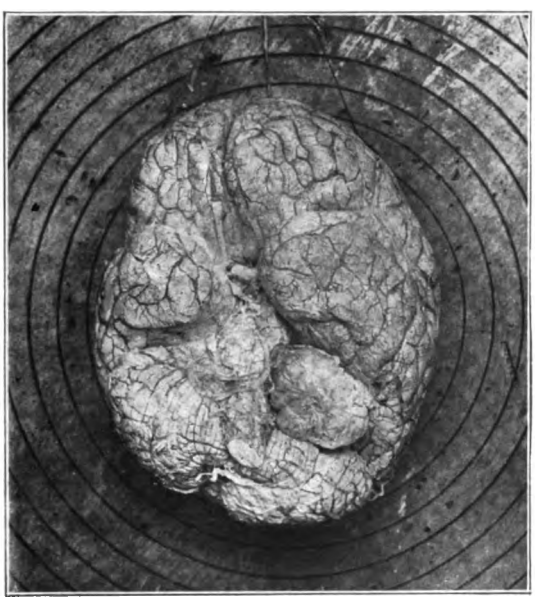

Fig. 2, Case 3.—Typical tumor of the left acoustic nerve in the left cerebello-pontine angle.

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weakness. Twenty-four hours after operation patient suddenly developed a typical tachycardia; pulse 240 beats per minute at heart; irregular, no radial pulse. This condition yielded to stimulation and subcutaneous infusions of salt solution, pulse returning to 108. The bilateral ankle-clonus disappeared, headache and vomiting ceased, and patient became comfortable.

Subsequent Course.—On third day paralysis of the pharynx was noticed, and up to his death patient was unable to swallow. On the fifth day patient had a convulsion, suddenly lost consciousness, became rigid all over, head thrown back in opisthotonus, radial pulse lost, tachycardia again persistent, respiration suspended; marked asphyxia. Patient recovered consciousness and seemed fairly well with the exception of the paralysis of the pharynx. In twenty-four hours he had a second attack of convulsions; this was followed by tachycardia and quick, shallow respiration. There was a ptosis of the right eyelid, loss of corneal reflex, corneal ulceration on the left eye; no loss of sensation in the left side of face. There was paralysis of the right arm and right leg, and perhaps of the left leg; death.

Autopsy.—This was made three hours after death. The cerebrum was dry and apparently completely drained of cerebrospinal fluid. There was more fluid below the tentorium cerebelli. On removing the tentorium, a tumor, the size of an English walnut, was found partly attached to the petrous portion of the temporal bone, loosely adherent, but apparently growing from the left acoustic nerve. It seemed so soft as to give the impression of being a cyst. The fifth nerve was stretched over the middle of the tumor, anteriorly. The entire cerebellum and brain stem seemed displaced, more especially the left lobe of the cerebellum, which was displaced downward and backward. The lower left half of the pons and adjacent portion of the medulla were markedly compressed (Fig. 2).

Death was evidently due to acute compression of the pons and medulla, brought about by the changing of the intracranial pressure, through the subtemporal decompressive operation. If a suboccipital decompression had been attempted on the left side, the tumor would have been found and could have been easily removed. Had the patient had any chance of leaving the table alive, the latter operation would have been attempted instead of the subtemporal.

The case also illustrates the difficulties in localizing even such a typical tumor as neoplasms of the acoustic nerve in the cerebello-pontine angle. Theoretically,

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this should be easy, but Bruns, Cushing and others have called attention to the absence of signs, and in this case up to within a few days of the operation there were practically no symptoms and signs which indicated with any degree of certainty that the growth was in the cerebello-pontine angle. I believe in our case the extreme softness, almost fluid consistency of the growth during life (although it became quite solid in formaldehyd solution), accounts for the absence of symptoms of pressure; the nerve trunks, being more firm than the tumor itself, were not compressed. Even the fifth nerve, which was stretched and flattened out over the tumor, showed no signs of loss of function, except twenty-four hours before death.

In this case we have typical history of a gradual diminution of hearing in left ear (side of lesion) beginning six years before the tumor caused general symptoms. Horizontal oscillations (jerking) of the eyeballs when turned toward side of lesion. No areflexia cornea at any period before the operation except a few days before death, although the autopsy showed that the fifth nerve was flattened into a ribbon over the tumor. There was no change in sensation, muscular power and reflexes in either upper or lower extremities. There was no choked disk until two months before the operation. In a word for six years after beginning deafness was noticed, no signs or symptoms of tumor of the cerebello-pontine angle were present. A few months before the operation, however, all general and focal signs rapidly set in. Then we have attacks, not continuous, of staggering gait; two attacks of astasia-abasia; inability to walk on account of static ataxia. Patient falls to the left; has no objective loss of power in arms or legs; perhaps a slight weakness of right leg; no ataxia of either arm; nystagmic jerking; weakness of internal rectus of right eye and external rectus of the left. Gait is not typically cerebellar; later on, weakness of right leg develops;

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bilateral ankle clonus; no ataxia in either upper or lower extremities when in recumbent position; no disturbance of sensation.

BASILAR MENINGITIS SIMULATING TUMOR

In this connection, I will give briefly the history of another case which I believe to be a case of basilar syphilitic meningitis, although there is no autopsy report to confirm the diagnosis. Both Weisenberg and Mills have placed similar cases on record. Oppenheim in a recent case report warns us not to place too much importance on a positive Wassermann reaction or on temporary success of the antisyphilitic treatment, in a similar case.

CASE 4.—*Patient.*—A woman, aged 47, married, four children, one miscarriage. Onset of present trouble in 1903, seven years ago. At that time, after having had shooting pains and general soreness of the legs for some weeks, she gradually lost power in both legs, the left one almost completely; the right one not so badly. She recovered after prolonged use of potassium iodid. She was well for five years.

March 22, 1910: Eighteen months ago the patient began to have defective vision, saw double, and began to lose hearing in the right ear; gait became uncertain; vomited often, but had no headache. Turning of the head caused vertigo, and standing erect or sitting caused vomiting. This condition remained unchanged. In the last six weeks prior to above date there had been a drooping of the left eyelid. There was intense pain at the base of the brain, and over the right eye. There were frequent spastic contractions of the right side of the face and the right shoulder-girdle.

Examination.—Mental condition and memory normal; bilateral choked disk; loss of corneal reflex of the right eye; weakness of the external rectus of right eye and of the internal rectus of the left eye. There was loss of hearing in the right ear, slight paresis of the right facial, no weakness of arms or legs; patellar reflexes exaggerated; ataxia of right leg in recumbent position; static ataxia; patient walked with uncertainty, legs widely separated; showed tendency to stagger and fall to the right.

Twenty-four hours after the examination patient was suddenly seized with an attack of acute bulbar paralysis and died within three hours.

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Here we have a typical syndrome of tumor of the cerebello-pontine angle: General symptoms of brain tumor plus one-sided involvement of the fifth, sixth, seventh and eighth nerves, with ataxia of one extremity on the same side.

The fact, however, that she had a paraplegia six years before which disappeared completely under the influence of potassium iodid, and that she died suddenly with all the signs of an acute bulbar palsy, lead me to believe that the case was one of basilar syphilitic meningitis. I did not recommend an operation, but placed her on gray oil injections, too late, however, to accomplish any results.

This case only strengthens the rule that a careful history and a consideration of the symptoms in the chronologic order of their occurrence is necessary to make the differential diagnosis.

EARLY DIAGNOSIS

I cannot lay too much stress on the early diagnosis of tumors of the cerebello-pontine angle. They are very amenable to surgical treatment, as the numerous cases on record testify; but when the case has advanced to the point of compression of the pons, as in my case, little is to be hoped for from surgical interference. The aurist should be the first to recognize these cases, because of the early involvement of hearing, and should be on the lookout for the early symptoms, viz., progressive loss of hearing with middle ear normal, vestibular attacks of vertigo, slight weakness of face and areflexia cornea on the same side, with perhaps weakness of external rectus of same eye. No other focal lesion, with the possible exception of basilar syphilis, produces this syndrome.

After the consideration of these cases we may say that tumors of the cerebello-pontine angle show a predominance of unilateral focal signs on part of the fifth, sixth, seventh and eighth, whereas tumors of the pons

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and medulla show bilateral focal signs with a clear indication of hemiplegia alternate. In both, the focal signs may precede the general signs of brain tumor, and frequently do. Pons and medulla tumors, being small, produce less marked general signs of brain tumor, whereas, the angle tumors have later on marked general signs. Choked disk is characterized by its absence in pons tumors, although this is not invariably the rule, whereas, the cerebello-pontine angle tumors manifest general signs. Choked disk is almost invariably present, together with headache, vomiting and vertigo, partly due to an associated internal hydrocephalus.

DIAGNOSIS OF CYSTS AND TUMORS OF CEREBELLUM

And now let us proceed to the consideration of that part of our subject which to my mind has the greatest practical importance, namely, the early diagnosis of cysts and tumors of the cerebellum itself. When these tumors and cysts have grown to such a size as to compress the cranial nerves and the pons itself, the diagnosis is not difficult, but the surgical prognosis is bad; whereas, the outlook is good if an early localizing diagnosis can be made before the tumor has grown beyond the confines of the cerebellum. This early diagnosis is very difficult. Not only is it difficult to say whether the growth is located on one side or the other, in the hemisphere, but it is often difficult to rule out the growths of the frontal lobe or to say with certainty whether the symptom complex is caused by a tumor, by acquired internal hydrocephalus, or whether we are dealing with the so-called pseudo-tumor symptom-complex.

CEREBELLAR CASES

Let me briefly introduce two cases here which will illustrate the difficulty of making an early exact localizing diagnosis in cerebellar cases:

CASE 5.—*Patient.*—J. P. C., Nashville, Tenn., a man aged 47, single; occupation, sheet-writer. Parents both dead; cause unknown. The patient had never had syphilis or a chancre;

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had been a drinking man for past ten years, but had never been sick. Onset of present illness eight months previous to examination. The chief symptom was a constant headache, usually occipital, but at times frontal. These headaches were very severe in character, and the periods of freedom were few and short in duration. Any physical strain, and also coughing, made the headache worse. When the headaches were bad the patient was confined to bed, but when he attempted to walk he staggered and showed a tendency to fall to the right.

Examination.—May 30, 1909. The pupils were equal in size, and responded to light. The eyeballs were prominent; external muscles of eyes normal; papillæ normal; corneal reflexes normal; hearing normal in both ears. Left nasolabial fold not so prominent as the right; no defect of function in regions supplied by any other cranial nerve. Muscular power, sensation and reflexes of both upper and lower extremities were normal. There was some static ataxia; marked ataxic gait; patient staggering toward the right.

In the course of the following six weeks there were no new developments, except that the headache and vomiting became worse, to the extent that they were very severe and almost constant. The gait became so ataxic that patient was not able to walk or stand without support. No choked disk developed.

Diagnosis.—Cerebellar tumor, probably of the right side.

Operation.—A suboccipital decompression on the right side was decided on, and the operation was performed by Dr. Horace Whitacre. No tumor was found. The cerebello-pontine angle was explored. The intracranial pressure was very great. About one-third of the right lobe of the cerebellum was removed.

Result.—The immediate result of the operation was a complete relief from headache and vomiting. There was some ataxia of the right hand after the operation. After two weeks the patient was able to sit up, and gradually began to walk. His gait, however, was decidedly ataxic. During the four weeks at the hospital, following the operation, he suffered but one headache. Five months after the operation his condition is fair, but he does not walk much better and has occasional attacks of headache.

CASE 6.—*Patient.*—M. M., a woman aged 36, was referred to me by Dr. F. Forchheimer on June 14, 1909. Patient was ill when an infant, but has since been in good health. Had influenza and whooping-cough four years ago. Had influenza nine weeks ago; since then constant headaches; partly frontal,

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partly occipital, the pain radiating into the shoulders. Had vomiting spells almost daily since the headache became constant. Had vertigo when the head was moved from side to side, or when asked to walk. Vision gradually failed, until patient was unable to read ordinary print.

Examination.—June 14, 1909. Facial expression one of great suffering; slight drooping of left eyelid; bilateral papilledema discovered by Dr. Walter Forchheimer one week ago; no loss of corneal reflexes; slight static ataxia when standing on right foot. The examination was otherwise negative.

During the next five weeks the condition gradually grew worse. Patient had repeated attacks of cerebellar spasms, during which there would be a momentary loss of consciousness, with general muscular rigidity. She also lost the patellar reflexes in both sides, and occasionally had double vision. There was no loss of muscular power in the legs ,but the ataxia became so marked that patient refused to walk, but was able to walk with assistance. The vision rapidly grew worse, and a suboccipital decompression was resolved on, the left side being chosen as the probable seat of the tumor, on account of the drooping of the left eyelid and a tendency to stagger toward the left.

Operation.—The operation was performed by Dr. Horace Whitacre. A cyst, about the size of an English walnut, was found at the seat of the operation. It was subcortical, involving the posterior portion of the cerebellum, and on opening it a pale amber-colored fluid was discharged, followed by a collapse of the lobe of the cerebellum. Recovery from shock and anesthesia was rapid.

Results.—Headache and vomiting ceased for a while, and the vision improved greatly, but the other signs remained unchanged. The choked disk continued, the double vision likewise. The patellar reflexes did not return. A sinus established itself automatically, and cerebrospinal fluid escaped in large quantities. After two weeks the headache and somnolence returned. There was an occasional attack of vomiting. There developed an acute ataxia of the left arm and leg, which rendered the patient unable to walk at all. There was also a marked weakness of the left arm. Incontinence of urine and feces developed, and there was a return of the cerebellar seizures. At this time I went away on my summer vacation and left the case in charge of Drs. Whitacre and Langdon. An attempt was made to relieve the patient by draining the lateral ventricles, but the patient died within twenty-four hours after the operation.

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TOPOGRAPHIC DIAGNOSIS OF CEREBELLAR TUMORS

The chief interest in the study of the differential topographic diagnosis of tumors below the tentorium cerebelli is whether we have arrived at that state of diagnostic acumen which will enable us to say with any degree of certainty whether a growth is located in the cerebellum itself, and above all, which lobe of the cerebellum is involved. I believe that we can say with some accuracy whether a growth is in the pons, or the region of the corpora quadrigemina, and I believe that the diagnosis of growths in the cerebro-pontine angle can be made with almost equal facility; but the great stumbling block is offered by tumors of the cerebellum itself. We have seen what signs and symptoms are peculiar to neoplasms of the pons, of the region of the corpora quadrigemina and of the cerebello-pontine angle.

In arriving at a diagnosis of tumors of the cerebellar hemisphere our first step is one of exclusion; we must exclude tumors in the above locations. Then we must exclude frontal lobe tumors. We cannot here go into the symptomotology of frontal lobe tumors, but suffice it to say that there are well known physiologic facts and clinical and surgical experiences which attest the fact that at times it is practically impossible to say whether the tumor is in the frontal lobe or in the cerebellum.

The physiologic fact to which I refer is that the loss or disturbance of function of one cerebellar hemisphere when removed in animal experiments, especially the ataxia, quickly disappears after the operation, but if the animal is then deprived of the opposite frontal lobe, all the cerebellar symptoms return. In a word, the frontal lobe in a measure takes up, or at least compensates for the loss of the function of the crossed cerebellar hemisphere. This interaction of function takes place through the fronto-bridge-cerebellar tract. It is also a well-known fact, that tumors of the frontal lobe, both by causing ataxia, and by producing distance pressure signs, can cause a symptom-complex, which at

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Fig. 3, Case 6.—Tumor of the right frontal lobe simulating a cerebellar symptom-complex.

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times can hardly be distinguished from a cerebellar symptom-complex. Moreover, it is not unusual to have unilateral spasms and even Jacksonian seizures in cerebellar lesions, as the cases of Mills and Weisenberg and Case 4 of this paper show, which complicates the matter still more.

FRONTAL TUMORS

Following is a short history of a case of left frontal tumor:

CASE 7.—*Patient.*—G. von L., aged 41, was admitted to Cincinnati Hospital Aug. 5, 1901, in a maniacal state, complaining of pain in the left occipital region. He denied venereal diseases and alcoholism. The patient was unable to give a history of his case. According to the wife's statement, he had failed in business six months previously; much worried. For the past six months she noticed a mental deterioration; he did not seem bright; was forgetful. For previous two months he had complained much of pain in the left occipital region. Six weeks before admission, while beating a carpet, patient suddenly staggered backward, was supported to a chair, stared at his wife, was unable to speak, became blue in the face, but did not lose consciousness. At the same time he had a spasm in the right leg, but none in the right arm. The patient said that he felt as if he were going up to the ceiling. He had about ten such attacks that night, three or four the next day and then they became less frequent and finally stopped. This was followed by formication and numbness of right foot. Later he had an attack of paralysis of the right side, which soon passed away and patient returned to his business. There were no headache or vertigo during this period. At times the patient would stagger and show a tendency to fall to the right side, and could not walk without support for the past few days. After a severe attack of pain in the left occipital region a few days before admission, patient had numbness and weakness of the right leg. He began to vomit and had attacks of vertigo. At night he became delirious and in a few days became maniacal and was brought to the hospital.

Examination.—Mental condition was bad; could be aroused from stupor, but was unable to give an account of himself; did not seem to understand the questions put to him; did not speak voluntarily; when asked a question, repeated it slowly, but gave no answer. Paresis of left facial was present; paresis of left side of tongue doubtful; patient was unable to state

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whether right hand was weak or not on account of mental condition; right leg was dragged when patient attempted to walk. Patient fell to right side. There was no ankle-clonus; no Babinski in right side. On ophthalmoscopic examination double choked disk was observed, with hemorrhagic retinitis; right pupil larger than the left; left pupil did not respond to light; right one responded. Slight ptosis of right eyelid; paresis of external rectus of both eyes.

Operation.—An unsuccessful operation was done over left cerebellar hemisphere. Patient died within thirty-six hours.

Autopsy.—Autopsy revealed a tumor the size of a hen's egg, which grew from the dura on the lower surface of the frontal lobe up into the frontal lobe. It was not infiltrating, and the frontal lobe surrounded the tumor like a shell.

This patient was operated on for a cerebellar growth chiefly owing to the fact that there was a hemiplegia alternans, involvement of the external recti of both sides and history of a staggering gait with tendency to fall to one side.

Only recently an autopsy was made in a similar case in which one feature of the clinical history was that the patient at times had a staggering gait. Dercum recently reported a similar case, where the operation was made over the cerebellum and the autopsy revealed fontal tumor. We are gradually, however, improving our knowledge of frontal growths, as well as of those of the cerebellum. I believe that we shall make fewer mistakes along these lines if we pay greater attention to the chronologic order of the development of signs and symptoms, and I believe that in frontal growths some change in the mental state will occur, and will often even antedate the general symptoms of brain tumor, whereas, mental symptoms if they occur at all, occur late in cerebellar tumors. In at least two cases of frontal tumors in my observation the mental deterioration was manifest, the above history showing that the patient at times even became maniacal, and was brought into the hospital in this condition. In the other case, which I refer to above, aside from a progressive loss of vision and the ataxia, there were no mental symptoms,

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except a remarkable euphoria, a cheerfulness which continued to death, lasting over four years, which is certainly unusual in brain tumor cases of other location.

Only recently Reich has shown that we may even have areflexia cornea, which is looked on as a postive diagnostic sign of tumors of the cerebello-pontine angle, in frontal tumors. This, however, is so rare that it need not complicate the diagnosis.

PSEUDOBRAIN TUMORS

In making the diagnosis of cerebellar growths, we must also rule out pseudobrain tumors, a subject which has been placed on record by Nonne, Oppenheim, myself and others. This pseudobrain tumor symptom-complex is more apt to resemble the cerebellar symptom-complex than any other. Its pathology is unknown. The autopsy in one of my cases was entirely negative, so far as the brain was concerned. Here is a summary of the typical syndrome: Headache, violent vertigo, vomiting, bilateral choked disk, paresis of the external recti muscles of the eyes, areflexia cornea, especially of the right eye; tinnitus, with diminished hearing on the same side, cerebellar ataxia and deflection of head and even entire body to one side. These signs of pseudotumor are in a manner similar to acquired internal hydrocephalus or serous meningitis. These two conditions must be ruled out, and the only means at our command are the presence of other signs and symptoms, on which I shall presently lay stress, which are present in cerebellar cases, and absent in the above symptom-complexes.

PHYSIOLOGY OF THE CEREBELLUM

A few words on the physiology of the cerebellum will be in place here. The chief function of the cerebellum as ascertained by animal experimentation, is that of automatic coordination. Through the medium of the cerebellar tract, Gowers' tract, the posterior column cerebellar tract, and the vestibular nerve, all of which Bruce

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holds end in the cortex of the vermiform lobe, the cerebellum obtains knowledge of the state of contraction, and the tone of all muscles of the body and the position of the body. When these tracts are interfered with within the cerebellum we have the chief symptom of cerebellar disease, viz., cerebellar ataxia. Lesions of the cerebellum, however, are not the only factor in the production of cerebellar ataxia. We know that tracts lead from the cerebellum to the pons through the corpora quadrigemina and to the opposite cerebral hemisphere, and that the destruction of any one of these tracts will cause cerebellar ataxia. Hence, lesions of the frontal lobe, of the corpora quadrigemina and of the pons may cause cerebellar ataxia. In the lesions of the cerebellum, however, we have ataxia without any associated signs, such as will form part of the syndrome in lesions of the frontal lobe of the corpora quadrigemina and of the pons. In considering cerebellar ataxia and other signs of cerebellar lesions we must not forget, that the loss of function of one cerebellar hemisphere, as Munk and others have shown, may be compensated by the opposite frontal lobe, and that symptoms may disappear; or that disease of one hemisphere may even run a latent course. Babinski lays much stress on a physiologic phenomenon which Munk attributes to the cerebellum, viz., a harmonious interaction between the movements of the extremities and those of the trunk. Babinski calls this function *synergie* and the loss of it *asynergie cerebelleuse*. By this is meant a loss of harmony, which shows itself when walking, for instance, by an attempt to bring the leg forward, while the trunk does not change its position or may even go backward. This symptom was typically illustrated by the tendency to retropulsion on attempting to walk in Case 1, or by the inability in Case 2, of the patient to walk forward when the eyes were closed. *Asynergie cerebelleuse* may be confined to one side—hemiasynergy—as in Case 6; and Babinski says that it points to a lesion of the cerebellar

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hemisphere on that side or to one of the tracts leading to the hemisphere, or from the hemisphere on that side. Oppenhim seems to agree to this; Spiller dissents. Forced movements are seen in animals, rolling movements, but Munk does not attribute them to lesions of the cerebellum, but to other causes. In the human being we see a tendency to assume certain positions in bed, or to hold the head to one side, the diagnostic value of which we will consider later.

Astasia abasia, hemiataxic movements, homolateral weakness of muscles, homolateral convulsions, will be considered as localizing signs later on, as will also speech disturbances and nystagmus, all of which have been attributed to loss of function of the cerebellum.

FOCAL SIGNS

What are the focal signs which clinical experience has taught us as pointing to tumors of the cerbellum? The following syndrome calls for analysis:

1. Cerebellar ataxia.
2. Atactic movements, hemiataxia, *asynergic cerebelleuse*, hemiasynergy and tremor.
3. Vertigo.
4. Nystagmic jerking and other eye-muscle signs.
5. Atonic paresis of trunk and extremity muscles on one side.
6. Adiadokokinesis and speech disturbances.
7. Convulsions, unilateral or bilateral.
8. Attitude of the head.

Let us take up the signs and symptoms in the order as they are given:

Cerebellar Ataxia.—The typical, reeling staggering gait is probably seen only in tumors of the vermiform lobe. Perhaps also in quadrigemina tumors (Case 1), but here it will be associated with extensive eye-palsies, which will be absent in the cerebellar lesion.

What is more commonly seen is staggering to one side and a tendency to fall to one side. I believe that when unassociated with pontine signs or symptoms, this

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may be looked on as fairly characteristic of lesions of the cerebellar hemisphere. It would be of great value, if it could be established as a law, that the patient reels or falls to the side of the lesion. Weisenburg seems to think that this is the case. Two of my patients showed a tendency to fall toward the side of the lesion; one did not. Oppenheim and Siemerling and Spiller hold that we cannot rely on this sign as pointing to the side of the lesion. Siemerling says that cerebellar ataxia may be entirely absent. If the vermis is the seat of the growth, the ataxia may be so great that the patient is unable to stand at all. This is true likewise of growths of the corpora quadrigemina, as Case 1 shows; but in advanced cases, as Nos. 3, 5 and 6 show, may be present in pressure in or on the lateral lobes, or in tumors of the brain stem. But this may be explained by distal or contiguous pressure on the vermiform lobe or cerebellar tracts, in all three of these cases.

Hemiataxia and Hemiasynergia.—These, which Oppenheim thinks may be identical manifestations of cerebellar incoordination, are often present, and may, after further investigation, be found to be of great topographic diagnostic importance as pointing to lesions in the same side of the cerebellum.

Bruns says that hemiataxia is a valuable sign, because it occurs early, and is a sign of an intracerebellar lesion. In the four cases reported by him it occurred in the homolateral arm in each case.

Soques reports hemiasynergy and adiadokokinesis on the same side, homolateral, in a case of tumor of the cerebello-pontine angle.

In Case 3, presumably a pons tumor, very marked hemiataxia, perhaps hemiasynergy, and certainly adiadokokinesis, developed four or five months after the onset of the first symptoms.

The homolateral ataxia which was seen in Case 5 after the operation, is in effect a physiologic observation.

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No tumor having been found, a large portion of the cerebellar hemisphere was removed, and the homolateral ataxia was noticed after a few days.

In Case 6 there was static ataxia of the right leg (side of cyst), before the operation, loss of patellar reflexes and atony of both legs, but no loss of muscular power. The ataxia became so marked that patient refused to attempt to walk. After the operation the ataxia of the left arm and leg became very acute. In a word, the cyst was found on the left side, and the ataxia was most marked on the left side, the right arm and leg being free. When ataxia is suspected but not present in the ordinary way of standing or walking, Oppenheim recommends that the patient be placed in the ordinary Romberg position, and be asked to bend forward with closed eyes. As a rule, not only will the loss of equilibrium show itself, but also the side toward which the patient sways, will show itself. Some stress has been laid on unilateral tremor of multiple sclerosis, as a sign of cerebellar disease. Collins, Bruns, Siemerling and Oppenheim have called attention to this sign, and when present, it is usually homolateral.

Atonic Paresis.—Atony of the trunk muscles on side of the lesion, homolateral weakness in the atactic limb and loss of patellar reflexes on one side or both, may occur, as seen in Case 6. Bruns says that it occurs only in acute cases after removal of a part of the cerebellum. Siemerling says that it is still a debatable sign. A sudden and acute onset of astasia abasia disappearing just as suddenly was seen in Case 3.

Vertigo.—This, which is invariably present in subtentorial growths, is not of much localizing value. The exact differentiation between vertigo of intra- and extracerebellar origin, as defined by Stewart and Holmes, has not been verified by others and needs further investigation, namely, that in intracerebellar tumors the ob-

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jects and the apparent movements of their own bodies are from the side of the lesion to the opposite side, and in extracerebellar tumors the apparent movement is toward the side of the lesion. If, however, vertigo is brought about by changing the position of the body or of the head, we may safely say that this occurs when the body or the head is turned away from the side of the lesion.

Nystagmus.—Not the typical nystagmus seen in multiple sclerosis, but a nystagmic jerking, when the eyes are turned to one side or the other, is frequently seen in subtentorial growths. Munk says that strabismus and nystagmus are not seen in animals as a result of the removal of one-half of the cerebellum, but as a result of injuries to the neighboring parts. Siemerling, although admitting it as a sign of intracerebellar lesion, thinks that it is caused by pressure on the pons or corpora quadrigemina. H. Neuman and Collins look on it as a cerebellar sign. Nystagmic jerking is usually seen when the eyes are turned toward the side of the lesion, but this is not the invariable rule. It was present toward the side of lesion in Case 3. It was not present in Case 5 either before or after the operation; nor was it present in Case 6.

Spiller, in a case of cerebellar abscess, says that the nystagmus became more marked when the patient looked toward the seat of the lesion. H. Neuman has conformed this in a later communication.

Siemerling does not feel that the direction of the nystagmus has a positive localizing value, while Oppenheim thinks that nystagmus may be a sign of intracerebellar disease, althought it more frequently is the result of distal pressure. I have never seen general ataxia of the eyeballs such as Spiller and Weisenburg describe in a subtentorial tumor, but have seen it in a case of nuclear degeneration of the centers in a case of chronic bulbar palsy.

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Attitude.—In this respect, I wish to call attention to the fact that the position of the head and the position of the body have a marked influence on bringing out signs of cerebellar tumors that may be latent. In Case 3 nystagmus occurred only when the eyes were turned toward the seat of the lesion. Oppenheim in a recent article gives his experiences on this subject. He cites two cases of tumor of the cerebellum in which a nystagmus was only slightly indicated when the patients, in a standing position, looked to one side or the other, but which become very marked even when the eyes were at rest, if the patients reclined on one side. In another case in which nystagmus was absent when standing or lying on the back, if the patient lay on his right side and looked toward the left, nystagmus appeared at once, and a paresis of the external rectus became apparent. In another case, nystagmus on looking toward the side of the lesion appeared only after the patient had been turned on a revolving chair, two or three times. In two other cases of subtentorial tumors, areflexia cornea was produced by having the patient lie on the side opposite to the tumor.

Convulsions.—General convulsions or unilateral convulsions, of the cerebellar type, viz., opisthotonos, momentary loss of consciousness with a general clonic spasm of very short duration, are indicative of subtentorial pressure and irritation of the pons, rather than of intracerebellar tumor. They were seen in Case 6 (cyst of hemisphere), as well as in Case 3 (tumor of cerebello-pontine angle). Probably one of the most valuable signs which will indicate the side of the lesion is one to which Babinski has called attention, namely:

Adiadokokinesia.—By this is meant a slowing of movements of an alternating character, and is best illustrated by successive pronation and supination of hand and forearm. In lesions of the cerebellar hemis-

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pheres there is a marked diminution in the rapidity with which these movements can be alternated, and occurs always on the homolateral side. Oppenheim and Collins and Siemerling lay much stress on this sign, and especially Siemerling, who says that he always found it after his attention was called to it.

Adiadokokinesia, according to Boenhoeffer, who describes speech disturbance in a case of cerebellar tumor, manifests itself as bradyphasia; he says that it is due to an inability of the patient to rearrange his lip and tongue muscles rapidly, which is necessary for fluent speech. Other speech disturbances have been ascribed to cerebellar lesions, as a staccato, jerky mode of talking, which has been attributed to incoördination or ataxia of the muscles of tongue, lip and larynx. Speech becomes slow, repetition of long words causes a chopped-off reproduction, and at times stumbling and stammering. Rapid speech is impossible, but there is no anarthria. The attitude of the head may give us some indication as to the side of the lesion. Baten called attention to the fact that the head is inclined in such a way that the occiput points to the side of the tumor, and the chin is pointed away from that side. Collins, in a case of hemorrhage, describes just the opposite condition. Oppenheim says that there is no rule, but that the patient assumes such a position of the head and body as cause him least inconvenience. Spiller also thinks that there is no rule. In my cases I could not confirm the rule. Sholz has used the Neisser puncture test to determine the side of lesion, and located the tumor in two out of three cases by this means. Oppenheim calls attention to a difference in the percussion note on the two sides, and says that we may find more dulness on the side of the lesion. Bruns calls attention, especially in children, to tenderness on percussion, and the cracked-pot sign, on the side of the lesion.

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BRAIN TUMOR OF PSYCHOMOTOR AREA

CAUSING JACKSONIAN AND GENERALIZED CONVULSIONS, VISUAL HALLUCINATIONS, SOMATIC DELUSIONS, ASTEREOGNOSIS AND HEMIPLEGIC PARALYSIS. OPERATION. RECOVERY, MENTAL AND PHYSICAL

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CINCINNATI

NEUROLOGIC REPORT BY DR. LANGDON

The case here reported presents no unusual features pertaining to the growth itself, its localization, the operative procedures or the recovery therefrom. In fact, it may fairly be classed as commonplace in those respects. It had been considered worthy of record, however, by reason of its peculiar symptom-complex, combining marked psychic as well as somatic disturbances; the practical absence of those important "general" symptoms; headache, vomiting, vertigo and papilledema; and the small amount of functional defect remaining at this date (March 16, 1910) four months after operation.

History.—Nov. 5, 1909, Dr. R. B. Hannah,[1] of Georgetown, Ohio, asked me to see a woman who had been developing some mental symptoms for a few weeks. It was also stated that preceding these evidences of insanity there had been some convulsive attacks, local and general. The development of the abnormal mental state, however, appeared to be the deter-

1. Acknowledgements are due and hereby tendered to Dr. Hannah for kindly contributing his personal notes and observations of the patient.

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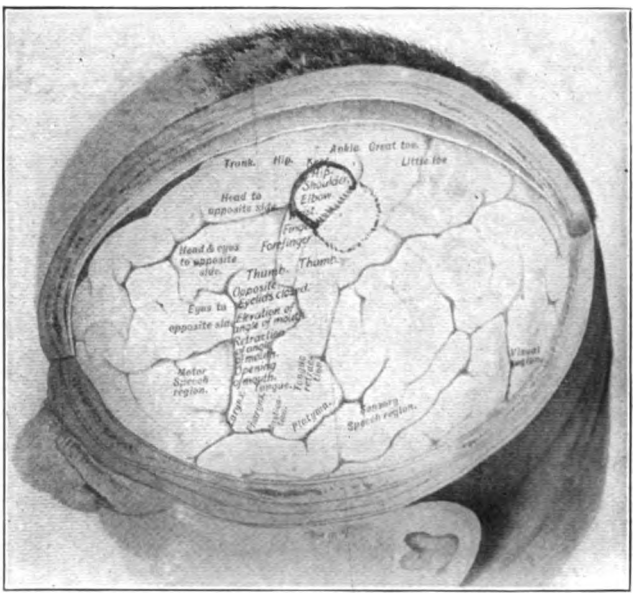

Fig. 1.—Location of brain tumor.

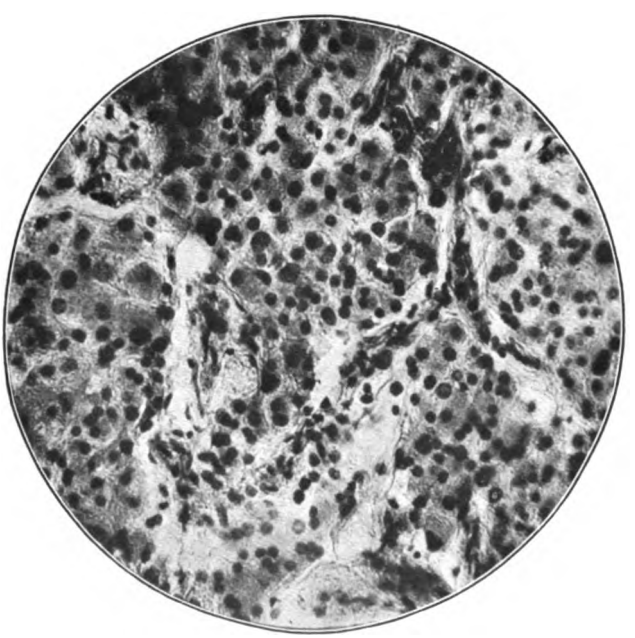

Fig. 2.—Photomicrograph of section of tumor (endothelioma).

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mining factor in seeking a consultation. Dr. Hannah had been in charge of the case a short time only, and at once recognized the probability of physical disease as a foundation for the nervous and mental manifestations.

The patient was a farmer's daughter, aged 39, single, intelligent and capable up to two years previous to my visit. She had for some years performed important clerical duties as a railway agent and telephone operator at a local station. The family history and surroundings were excellent and prosperous. There was a history of a blow with a croquet mallet on the left parietal region in childhood. There were no sequelæ of importance following this injury; the patient was not unconscious and not confined to the house on account of it. There was no scar at the alleged site of the blow. Two years previous to this examination there had been a "jerking" of the right hand and arm, followed in a few moments by "unconsciousness." These attacks had been repeated about twenty times in the two years just past. Following them was a progressively increasing weakness of the right hand. Two weeks previous to this examination the patient had visual hallucinations and some delusions that surgical operations had been made on her eyes and right leg.

First Examination.—This was made at the home of the patient Nov. 5, 1909. The patient was in bed. Consciousness was somewhat clouded, but the patient carried on ordinary conversation fairly well. She appeared mildly elated, or perhaps a state of euphoria would better describe the emotional state. Asked if she sees anything unusual, she answered "Yes; holes in the ceiling and walls; small ones; many of them." There was no defect in ceiling, nor was it papered. There was nothing to be mistaken for holes; in other words, this was a true hallucination, not an illusion. While the pupillary reactions were being looked for, she volunteered the information that her "eyes had been operated on recently" (probably the opthalmoscopic examination made a few days previously). Asked if anything else had been done, she replied: "Yes, my right leg was cut off recently." This was stated in quite a matter-of-fact manner, without any evidence of regret or emotion. The patient conversed rationally on many ordinary topics, but on being questioned as to any speech defect, she said she "loses a word occasionally." Speech tests showed reception and understanding good for ordinary words. Test-words and sentences were repeated correctly and without defect of articulation. Internal speed appeared good; she could describe objects, letters, words, etc., and tell their meanings.

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With the practically powerless right hand she could tell the shape of objects, but could not name them (astereognosis). Owing to weakness in this hand, she could not grasp a pencil.

Cranial Nerves: I, not tested; II, ordinary vision, good; no fundus changes reported by Dr. H. P. Shelton, who kindly examined the eyes a few days before my visit; III, IV, VI, no ocular palsies; pupils reacted well to light and accommodation; V, no defects noted; VII, slight weakness in lower right face group; VIII, hearing good to ordinary tests (voice and watch); IX, X, XI, no defects noted; XII, tongue movements good; no trophic changes.

Trunk and Extremities: Motion—Right hand and entire arm useless, relaxed; could bend elbow and wrist and close fingers, but had no grasp; patient could not hold pencil; right leg also weak; could move joints, but could not walk; power in left arm, hand, leg and foot appeared good.

Sensation: No anesthesia to ordinary touch and pin-prick; no differences noted between the two sides of body and limbs.

Reflexes: Organic—Not defective. Tendinous—Knee-jerks diminished, but distinctly present. No difference in the two sides. No spasticity. Cutaneous—Plantar; no response, or occasionally a flexor response; no difference in the two sides.

Trophic symptoms absent; vaso-motor symptoms absent.

Diagnosis.—Irritative lesion of the left psychomotor area, probably tumor; possibly a cyst or abscess.

Removal to hospital for further observation and probable operation was advised and the patient was taken to Cincinnati on Nov. 9, 1910.

Second Examination.—Nov. 10, 1910, Ophthalmoscopic examination by Drs. Robert Sattler and Victor Ray revealed no changes in the fundus. Blood examination by Dr. W. E. Schenck, on the same day, showed reds, 4.460.000; whites, 3.600; hemoglobin, 65 per cent.; color index, 0.74.

Differential count: Polymorphonuclear, 81; mononuclear (small) 10, large 5; eosinophiles, 4; total, 100.

Diagnosis.—Secondary anemia.

Uranalysis (by Dr. H. L. Woodward): Color, amber; reaction, acid. Albumin, trace (probably due to fecal contamination; disappeared later).

OPERATION REPORT BY DR. KRAMER

First Operation.—Nov. 13, 1909, under ether anesthesia, an opening was made in the skull over the upper half of the left Rolandic area, two inches in diameter. There was no bulging

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of the dura and no pulsation visible. In the center of the operative field the dura was flattened and darker in appearance. Palpation revealed the presence of a circumscribed mass, harder in consistence than the surrounding cortex. The patient was not very strong before the operation and, as a result of the cranial resection there developed considerable shock. For this reason, the scalp was sutured and the removal of the tumor was delayed for a second operation, after the method of Sir Victor Horsley. After an interval of ten days the patient improved markedly in strength and on Nov. 23, 1909, the second operation was done.

Second Operation.—On opening the dura, we found a tumor imbedded in the brain, adherent to the pia mater, involving principally the precentral convolution and the Rolandic fissure (Fig. 1).

The tumor was easily lifted from its bed in the brain, after severing a few slight adhesions to the pia mater. Hemorrhage was insignificant and there was but little shock. At the time of the second operation the brain pulsated very plainly as the result of the previous decompression. The tumor occupied the middle third of the left precentral convolution, growing downward and diagonally backward across the Rolandic fissure and displacing, but not invading, the postcentral convolution. The long axis of the tumor (55 mm.) ran across the Rolandic fissure, downward and backward.

Examination of Tumor.—The tumor was encapsulated, firm and resistant on palpation, presenting numerous small protrusions and cysts on its buried surface. It was not adherent to the dura mater. The tumor weighed 26 gm. and on section proved to be an endothelioma springing from the pia mater (Fig. 2).

The recovery from the surgical procedure was uneventful and the patient returned to her home eight days after the second operation.

Postoperative Notes (L).—Patient put to bed in good condition, conscious; improvement was steady. The hallucinations and delusions had disappeared on the second day. There was no "jerking" in the face, arm or leg. On the eighth day patient could move the paralyzed (right) leg, but could not stand alone. She could move the fingers of right hand, but had no use of it for any purpose. She was removed to her home (forty miles) on a traction car at this time. After arrival home she sat up daily in her chair. Dec. 20, 1909, twenty-seven days after final operation, she took a few steps,

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with assistance. Jan. 1, 1910, she walked alone across the room.

April 7, 1910, 4½ months after operation, she traveled forty miles on a traction car and called at my office for reexamination, when the following notes were made:

Height, 5 feet 1½ inches; weight, 96 pounds; no mental defect; no speech defect.

Motion: No "Jacksonian attacks," convulsions or unconscious attacks since the operation. Walks well; no defect of gait or station.

Power: Can close right hand, but cannot "register" on dynamometer (standard). Grasp in left hand registers 20 K. Moves right upper extremity freely at all joints, but has little use of hand; cannot oppose the thumb and fingers or make a "fist."

Some "silver-fork aspect" of right hand and wrist. The right shoulder droops slightly. The left sternomastoid muscle is weak, but competent in turning head.

Sensation: Good for tact, pain, heat and cold, everywhere in both hands and arms. Astereognosis *complete* of right hand, front and back, extending half way up forearm. Cannot recognize any difference between a pocket knife, finger, coin or wisp of cotton with hand. Half way up forearm on flexor surface she recognizes a silver half dollar as "hard and flat." Some sensory defect in sole of right foot, where cotton and a finger touch feel "the same," but recognizes a touch with a "stick" (toothpick). "Tickle sense" good in both feet.

Reflexes: Organic—No impairment. Tendinous—Knee-jerks slightly plus and equal. Ankle-clonus absent on both sides. Cutaneous—Plantar reflexes, flexor; the right less marked than the left. Oppenheim reflex absent.

Patient goes about the house and grounds daily and attends to personal needs so far as the defective right hand permits. She feels that she is gaining steadily and is cheerful and hopeful generally.

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THREE CASES OF TUMOR IN THE CEREBELLO-PONTILE ANGLE

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CHICAGO

Since the subject of tumor of the brain has ceased to have a mere academic interest, and has become the coveted point of attack of every progressive surgeon, reports of well-observed cases have become regular topics for discussion in neurologic and surgical gatherings. From the point of view of successful removal, next to neoplasms situated in the Rolandic area, tumors growing in the angle between cerebellum and pons offer the greatest possibilities for success, and this for two reasons: First, thanks to the labors of modern physiologists and clinicians, the localization of tumors in this region has become comparatively easy; second, the surgical technic has reached a high degree of perfection. When, therefore, a diagnosis can be made early and a skilled surgeon is at hand, a perfect result should be obtained in those cases in which the tumor is at all removable. Nevertheless there will always remain a large number in which removal is impossible, either because the tumor is too large, or because it belongs to the infiltrating variety.

Being of the opinion that our failures are sometimes more interesting than our successes, and also that statistics which exclude unsuccessful cases are fallacious, I

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am reporting three cases of tumor in the cerebello-pontile angle in which the diagnosis was correct, but which were nevertheless failures, if by success we mean with Charles K. Mills not only exact localization, but also the successful removal of a tumor, and at least partial success as regards removal of the disease.

As the entire subject of cerebellar and cerebello-pontile angle tumors has been ably reviewed by Mills, Frazier, Weisenburg and Frenkel,[1] also by Harvey Cushing,[2] and quite recently by Risien Russell,[3] I refer the reader to these authors for a complete account of the varied symptomatology of these neoplasms.

The interest in my report being centered in the cases themselves, I shall follow each case with some general remarks:

CASE 1.—*Patient.*—A. B., aged 24, single, clerk, with negative family and personal history, was first seen by me Dec. 28, 1905. His brother, a physician, who accompanied him, stated that, though physically always in good health, the patient has been considered nervous since childhood. At an early age he had suffered from night-terrors and even at the time of examination he regularly appeared dazed and confused on awaking in the morning. He was a moderate drinker and smoker and had never acquired syphilis.

Present Illness.—In August, 1905, the patient felt a weakness in the left leg and somewhat later in the left arm; the face was not involved. In the following September he began to suffer from occasional headaches of moderate severity, which were always diurnal, aggravated by physical exercise and localized in the frontal region and the nape of the neck. At about the same time transient attacks of dizziness appeared whenever he rose suddenly from the recumbent posture. Soon thereafter vision began to fail: the eyes were blurred and attacks of blindness occurred, which, on passing off, left black spots in his visual fields. Accompanying the disturbances of sight were attacks of dizziness, but never nausea or vomiting. Of irritative motor phenomena the patient described peculiar jerkings and twitchings of the chest muscles, especially marked during

1. New York Med. Jour., Feb. 11, 18, and 25, 1905.
2. Cushing, Harvey: Boston Med. and Surg. Jour., clxi, No. 31, p. 71; Interstate Med. Jour., September, 1909.
3. Russell, Risien: Brit. Med. Jour., Feb. 19 and 26, and March 5, 1910.

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excitement. During the month preceding this examination he had been almost constantly tired; the slightest exertion brought on extreme fatigue and sleep was not refreshing; vision had been failing rapidly and the gait showed some staggering to the left. While these symptoms were developing two ophthalmologists were consulted. Each discovered a papilledema and suggested neurologic treatment. The next physician in attendance pronounced the case a functional neurosis. The brother, not satisfied with the diagnosis, brought the patient to my office Dec. 28, 1905, about a month after the previous consultations.

Examination.—This revealed a well-nourished young man of average weight and height, whose viscera were normal, pulse regular and 72 beats per minute. Paralysis of any kind was not apparent. Though the left half of the face seemed somewhat relaxed while at rest, yet when the patient was speaking and performing the usual motor tests nothing abnormal could be detected. The left hand-grasp was somewhat weaker than the right and the feft leg showed a slight degree of weakness when made to move against resistance. The gait was somewhat uncertain and slightly ataxic, with a tendency to reel more often to the left than to the right.

Cranial Nerves.—The olfactory nerves were not involved. The optic nerves showed choked disk and beginning atrophy, in consequence of which vision was markedly reduced. The pupils responded somewhat sluggishly both to light and in accommodation. Nystagmoid jerking appeared when the eyes were directed in extreme lateral positions. The third, fourth and sixth nerves were not involved. When first examined patient made no complaint of paresthesia in the region of the left trigeminus, nor was anything found objectively. Two months later, however, corneal areflexia was distinct and analgesia pronounced in the same region. With the exception of a slight degree of relaxation the left seventh nerve appeared normal. A systematic examination disclosed the existence of nerve-deafness unknown to the patient and limited to the left side. The cranial nerves from the ninth to the twelfth, inclusive, were not in the least affected.

Reflexes. —All the superficial reflexes were present and of about normal intensity. The deep reflexes in upper and lower extremities were markedly exaggerated, more so on the left side; ankle clonus, Babinski, Oppenheim, or Gordon signs were not obtainable.

Coordination.—Slight static and dynamic ataxia was present in the lower extremities. When each leg was tested separately the greater degree of incoordination was found in the left. The

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finger-to-nose test showed marked tremor in the left hand—more of an ataxic than an intention tremor. Cerebellar asynergy in trunk and lower extremities was absent, likewise Babinski's adiadocokinesia sign.

Diagnosis and Treatment.—My diagnosis was tumor in the left cerebello-pontile angle. Immediate operation was recommended with the expectation of saving what was left of patient's vision. The brother having charge of the case successively engaged three surgeons, who performed a number operations within a period of about two months. The first surgeon trephined over the left cerebellum; but, on discovering the presence of pulsation and no tumor, he replaced the bone and closed the wound. One week later the same surgeon attempted a ventricular tapping, but found no fluid. About two weeks later another surgeon was consulted, who also trephined over the left cerebellum. After an unsuccessful search in the region of the left angle he concluded the operation by decompressing the right subtemporal area. Meanwhile the patient had become completely blind. This circumstance caused me to discourage further operative undertakings. Nevertheless, a third surgeon was found who reluctantly yielded to the brother's request to make a new hunt for the elusive tumor. Again the left cerebellum was uncovered and the left cerebello-pontile angle explored. The operation was a bloody one and lasted several hours. In spite of a profuse hemorrhage, which dictated a discontiuance of the operation, the surgeon was urged on to continue at any cost. At last his persistence was rewarded by finding a neurofibroma, which he thought probably originated in the sheath of the left acoustic nerve. Because of the patient's precarious condition it was possible only to remove a part of the tumor, namely, a piece the size of a cherry. The surgeon concluded the operation by making subtentorial decompression over the left cerebellum.

Subsequent History.—Recovery from the operation was uneventful and the patient became free from headache and dizziness, but the tumor symptoms were even more numerous and more pronounced. To mention them only, there were: adiadocokinesia, reeling gait, left-sided motor weakness, optic atrophy, left corneal areflexia, complete paralysis of the sensory trigeminus and left-sided deafness. The patient's condition remained practically the same until the middle of September, 1909, when severe headaches made their appearance. These resisted all forms of medication, and surgical treatment was again considered. Dr. Allen B. Kanavel, with whom consultation was had, favored a cautiously performed lumbar puncture rather than a ventricular tapping, being fully aware that such procedure has been condemned in tumors of the posterior fossæ.

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A small quantity of spinal fluid was removed, but this sufficed to give immediate relief from headache; the patient was again able to be about as before. Unfortunately this remission was of but short duration, for the end came unexpectedly on Dec. 2, 1909. While the patient was conversing over the telephone he had a peculiar seizure, in which he fell backward, striking the ground with his head and becoming unconscious. Coma supervened and he died within two hours after the fall.

Post-mortem Examination.—Owing to delay in obtaining permission, the brain could not be examined until eighteen hours after death, which, of course, was fatal to the application of the finer neuroglia stains. Macroscopically the brain presented two hernial protrusions: one situated over the right subtemporal region, probably the result of a decompression operation; the other hernia was seen over the left cerebellum, the site of several operations. In my attempt to remove the brain from the cranial cavity I ruptured a cyst containing about six ounces of fluid blood, evidently a recent hemorrhage. The cyst-wall was adherent to the petrous portion of the left temporal bone and constituted a part of the hernial protrusion over the left cerebellum. After hardening the brain in formalin solution neurologic frontal sections were made. With the exception of bilateral internal hydrocephalus, more marked on the right side, nothing pathologic was discovered in the cerebrum. The hydrocephalus was probably the result of obliteration of the foramen of Magendie and of the foramina of Luschka. The conspicuous pathology was in the cerebellum. The entire left hemisphere was occupied by one large mass of denser consistency, but not of different configuration, from the normal cerebellum, encroaching somewhat on the structures of the right half (see illustration 1). A horizontal section through the entire left half of the cerebellum revealed a surface of mottled appearance, strongly suggestive of old and new hemorrhages. Microscopically the sections showed parts in which dense networks of fibrillar ground-substance were in close relation with numerous polymorphous cellular elements, varying in size from the small round cell to the long spindle-shaped cell, each containing a granular nucleus. In other parts of the sections could be seen islets of round cells of varying size with scarcely any intervening intercellular substance. In addition there were noticeable throughout the tumor mass numerous vessels of varying caliber. The mottled appearance, previously observed with the naked eye, under the microscope proved to be irregularly scattered hemorrhages of various sizes and ages. For the miscroscopic sections I am greatly indebted to Dr. C. D. Hauch, and also Prof. F. R. Zeit, in whose laboratory they were prepared.

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Pathologic Diagnosis.—Because of the presence of areas resembling sarcoma, and of others corresponding to the appearances of glioma, the diagnosis presented difficulties. There are those who call such tumors gliosarcomata, while others think that this term is merely a cloak for our ignorance. It cannot be denied that rarely both epiblastic and mesoblastic formations may occur in the same tumor, but I incline to the view that most of those reported as gliosarcomata have been either gliomata or sarcomata. It is generally believed that glioma in its varied metamorphosis is capable of producing pictures like sarcoma. The preponderating appearance of my specimen was like that found in glioma; besides, the growth did not overstep physiologic and anatomical boundaries. My diagnosis was glioma cerebelli. In the discussion which ensued on my presentation of the brain with microscopic sections in the joint meeting of the Chicago Pathological and the Chicago Neurological Society, this diagnosis received substantial support.

COMMENT

It is interesting to note that in this, as in many another neurologic case, the ophthalmologists were the first to call attention to the brain as the probable seat of the lesion in a supposed affection of the eye; further, that neurasthenic symptoms had at one period so overtopped the underlying basic trouble that an experienced physician had made the diagnosis of neurasthenia four weeks prior to my seeing the patient, at a time when tumor symptoms must have been present. Another point worth noting is the unusual beginning of the trouble with weakness in the left leg and arm, a weakness which corresponded to the asthenia of Luciani, and was not the result of a motor paralysis, there having been neither disability nor spasticity and pathologic reflexes. The case began, then, with asthenia confined to the left side, without any known cranial nerve disturbances—unless nerve-deafness had existed for some time, of which the patient had no knowledge. Then followed headache, dizziness, and visual disturbances, all general symptoms of brain tumor. Later still came cerebellar incoordination and only after several operations had been performed was trigeminus anesthesia noted.

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A retrospect of the surgery in this case brings out the following important points:

1. The presence of pulsation does not necessarily mean an absence of tumor underneath.

2. Once a tumor patient has become blind and his other symptoms, such as severe headache, vomiting and vertigo, are not troublesome, a radical operation may possibly verify a doubtful diagnosis, but is of no service to the patient.

3. A knowledge of the previous point should lead to the performance of "decompression" before blindness has occurred.

4. The much-dreaded lumbar puncture in tumors of the posterior fossæ may occasionally prove beneficial in the hands of those who exercise due care in preventing a sudden escape of the spinal fluid. In this case the removal of only about an ounce of cerebrospinal fluid was sufficient to relieve a headache which resisted all other treatment.

In respect to the discrepancies existing between the ante-mortem pathology, which showed a neurofibroma, and the post-mortem findings, which revealed a glioma, two possibilities must be considered. Either the glioma, which, it will be recalled, contained areas resembling sarcoma, developed since the operation and grew into a previously existing sarcoma—the latter a malignant transformation of remaining fibroma tissue—or else the portion of tumor removed at operation was not a fibroma. Not having seen the first specimen, but having carefully examined the post-mortem sections, I incline to the view that the tumor was a glioma from the beginning.

Equally uncertain is the exact origin of the hemorrhagic cyst. It is quite possible that the fall which occurred when the patient was standing at the telephone was entirely accidental and the cause of the large hemorrhage which produced death by suddenly compressing the medulla. It is more probable, however, that the

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large hemorrhage was spontaneous and orginated in the same way as did the other numerous large and small hemorrhages, which were not sufficiently extensive to press on the pons-medulla and thereby to cause death. Neither the macroscopic nor the microscopic examination, however, threw any light on this question.

As for the internal hydrocephalus, its occurrence has been frequently noted in tumors of the cerebellum and the cerebello-pontile angle, and has been attributed either to agglutination of the foramina connecting the fourth ventricle with the subarachnoid lymph-spaces, or else to the compression by the tumor of the veins of Galen, preventing drainage of the interior.

CASE 2.—*Patient.*—Miss A. C., aged 26, bookkeeper, consulted Dr. R. C. Pattillo in the spring of 1905 for an affection of the eyes. He discovered a refractive error requiring correction with lenses. These having been prescribed, the patient was placed in the care of Dr. Frederick Menge for the treatment of peculiar sensory troubles in the face, presumably caused by antrum disease. Repeated examinations by Dr. Menge failed to disclose the existence of either antrum or sinus disease. The patient was returned to Dr. Pattillo, who made several ophthalmoscopic examinations, and, finding the optic disks normal and no other ophthalmic lesion, concluded that the case required neurologic treatment. Through his courtesy I saw the patient for the first time in July, 1905, and obtained the following history:

History.—The family record, though not of the best, was free from nervous and mental disease, nor could I discover a history of tuberculosis or neoplasm. The patient, never very robust, had always been well until added responsibilities had of late caused a "nervous breakdown." She stated that within the past three months she had developed attacks of sudden blindness, which were momentary in duration and most often occurred when she was recumbent. Though these symptoms at first alarmed her, she continued at work until more troublesome visual disturbances appeared in the form of mists or clouds rising before her eyes, which temporarily obscured her vision while at her books. After a series of these attacks she was obliged to take a few days' rest, but she regularly returned to her work thereafter. A complaint to which she paid little heed, but which had persisted several months before the eye symptoms became prominent, was a peculiar heaviness and

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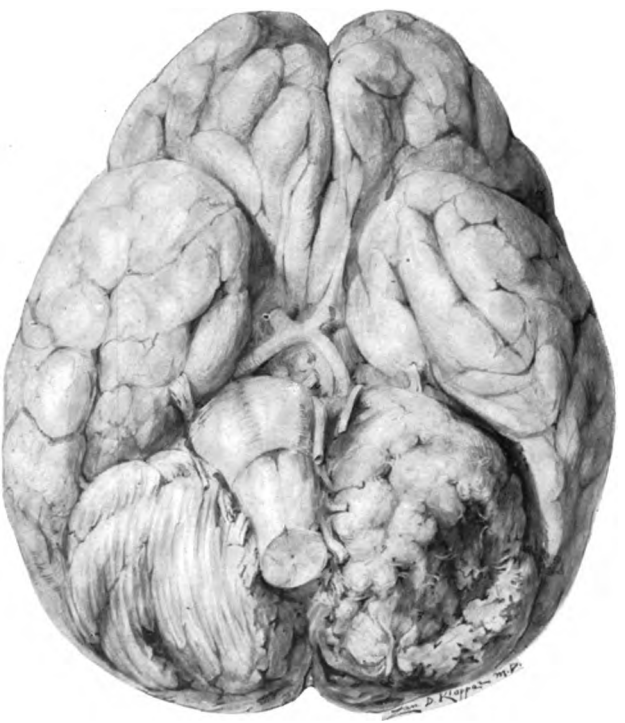

Enormously enlarged left half of cerebellum, pushing pons-medulla over to the right and containing large glioma.

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pressure feeling, with an occasional sharp pain in the left cheek, often radiating into the left eye. Not infrequently she experienced sensations of "grittiness" in both eyes, followed as a rule by an irresistible desire to sleep. While eliciting this history I discovered marked deafness in the patient's left ear, which, she explained, had come on gradually after more or less constant ringing had persisted in that ear for several months. And finally she laid particular stress on the existence of a feeling of fatigue, especially on rising in the morning, complained of general nervousness and dull headaches, and also of occasional numbness in the finger tips.

Examination.—The patient, an intelligent young woman, of about 26, appearing rather nervous and somewhat depressed, recited her story in great detail and gave it the coloring of the hysterical. A physical examination disclosed normal viscera. Motor paralysis was nowhere discernible, nor could twitching of muscles be seen anywhere. Gait and station were normal. The sensory examination revealed slight hypesthesia over the entire left half of body, including the face. Superficial and deep reflexes were all normal, with the sole exception of some reduction of the conjunctival and corneal reflexes on the left side. The pupils responded well to light and in accommodation. The optic disks were neither congested nor pale; in fact, they appeared typically normal. Hearing was completely abolished in the left and normal in the right ear. Additional findings were: A point of tenderness over the vertex, several such points over the spine and a hypersensitive spot in the left ovarian region. As the symptoms, both positive and negative, were those frequently encountered in functional nervous disease, and as none of the objective signs appeared to me definitely characteristic of organic disease, I diagnosed hysteria and treated the patient accordingly. She visited me several times, seemingly improved at first, but not so during the last two or three visits.

Subsequent History.—In the middle of September, 1905—six weeks after my first examination—the patient entered my office staggering and in a state of alarm. With great consternation she related to me that, as she was crossing the street, she became dizzy, staggered, and everything appeared black to her. It was marvellous, she said, that she escaped being killed, having become blind while crossing the busy thoroughfare. Her sight had gradually returned, but she feared a recurrence of the attack. She also informed me that two days previously she had suffered intense headache, had developed dizziness and had vomited several times. Never before had the last-mentioned symptoms been in evidence.

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Her gait had become ataxic—a reeling from side to side. The ophthalmoscopic examination revealed to me, for the first time in this case, extreme bilateral choking of the disk.

Diagnosis.—The advent of such symptoms compelled a revision of the diagnosis. Everything suddenly appeared clear to me: The deafness, having come without antecedent local cause and having been preceded for months by tinnitus aurium, could now be readily explained by pressure on the acusticus. And similarly, the paresthetic disorder in the left half of the face, which had given rise to the suspicion of antrum disease, could easily be ascribed to pressure on the trigeminus. The frequent and peculiar visual disturbances, in spite of the presence of normal disks in the early history of the case, could be accounted for by transient attacks of edema. Of course, with the appearance of headache, vomiting and vertigo, added to the cerebellar gait, in a patient who had had acusticus and trigeminus involvement, the diagnosis of tumor in the cerebello-pontile angle on the side of the cranial nerve lesions became almost a certainty.

With this diagnosis and the advice to operate immediately, the patient was returned to her family physician. Two weeks later a two-step operation was performed over the cerebellum, during the progress of which the diagnosis was verified, but the patient died. Consent was not obtained for a post-mortem examination.

COMMENT

In this, as in the preceding case, eye symptoms were among the earliest to attract attention. Paresthesia in the left trigeminus, though antedating the visual disturbances by several months, failed to impress the patient with its seriousness. Likewise, a deafness which was complete—discovered during a routine examination—barely received mention on the part of the patient. To her the visual and neurasthenoid symptoms were of far greater importance, and by manner and statement she succeeded in impressing her physician with a similar belief. This may perhaps explain why manifestations which were unmistakable local signs of tumor in the cerebello-pontile angle, did not receive due consideration until the general symptoms of brain tumor appeared. Further, this case helps to support the following conclusions:

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1. Unilateral sensory disturbances, be they ever so slight, should arouse a suspicion of organic disease, because these are not invariably the result of functional neurosis.

2. The focal signs of neoplasm of the brain, and particularly of that growing in the cerebello-pontile recess, often precede the general symptoms by a considerable period of time.

3. Choked disc may occur late, but rapidly, in tumors of the posterior fossæ.

CASE 3.—*Patient.*—C. T., female, aged 55, entered Cook County Hospital on Nov. 26, 1909, in the service of Dr. G. W. Hall, by whose courtesy I am reporting this case. A few days after her admission I made an examination and learned that the patient, whose family and personal history were negative, had been well until about six months previous. At that time she developed dull headaches, which came at irregular intervals, each attack usually lasting several hours. Their localization was principally over the right fronto-parietal region and occasionally over the occiput. Soon after this symptom there appeared more or less constant dizziness, which was always aggravated by the upright posture and by sudden rising from recumbency. Following this, came ataxia of a type which caused the patient, unless supported, to reel and fall mostly to the right, but occasionally to the left and forward. The aforementioned symptoms were rapidly succeeded by attacks of nausea and projectile vomiting having no relation whatever to the ingestion of food. From the beginning of her illness vision had been gradually failing, but she could still read newspapers six weeks before admission; from that time on her sight had deteriorated rapidly, so that at this examination she could only count fingers. An additional complaint was, that six months previously she had developed what was considered a "stroke" on the right side, which left some weakness on the same side.

Examination.—The patient, a well-nourished woman of about 55 years, walked slowly, with short steps, her gait presenting a good example of the intoxicated, but not that of one suffering from past or present motor paralysis. The facial musculature appeared somewhat relaxed on the right side, although on inspection no definite paralysis was detectable. On the application of the usual tests I discovered no noticeable inequality in wrinkling the forehead, but winking was less frequent on the right than on the left side; further,

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in a voluntary attempt to raise the upper lip, the right half was somewhat retarded, and puckering of the lips as well as whistling seemed impossible. The tongue, when protruded, deviated markedly to the right and the mucous membrane of the right half was thrown into folds, giving the typical appearance of lingual hemiatrophy. With the exception of distinct paralysis of the right external rectus, the ocular muscles showed no anomalies beyond a slight nystagmus, slow in character and most marked when patient looked to the extreme right. The pupils were dilated and responded but faintly to light and accommodation. Vision was reduced to finger-counting. Involvement of the trigeminus was manifest by the presence of hypesthesia and hypalgesia in its entire distribution, including cornea, conjunctiva, nasal and oral mucous membranes. Hearing was entirely abolished in the right and slightly below normal in the left ear. At this point it is well to state that about twelve weeks before entrance an incision in the right external auditory meatus had been made, which resulted in a chronic purulent discharge from that ear. The reason for such treatment was not ascertainable.

Reflexes: The corneal and conjunctival reflexes were absent on the right, present on the left side. Epigastric and abdominal reflexes were not elicitable on either side. Masseter, supinator, biceps, triceps, knee and Achilles tendon reflexes were present bilaterally and were of normal intensity.

Sensation: This was nowhere impaired, except in the right trigeminus distribution.

Coordination: There was undoubted incoordination of both station and locomotion of the variety described as cerebellar: a tendency to fall sidewise when standing or walking, which was not aggravated by closure of the eyes. The cerebellar symptom described by Babinski under the term adiadocokinesia was present and well-marked; the patient was unable alternately to pronate and supinate the right forearm, but could accomplish these movements readily with the left.

The viscera were all normal; likewise the blood and urine, which were frequently examined. A Wassermann test, kindly made for us by Dr. Frederick Harris, proved negative.

Diagnosis.—This was easy in the presence of the many general and local symptoms of brain pressure as found in our case. In addition to the general symptoms of brain tumor, such as headache, nausea and vomiting, bilateral choked disk, there was nystagmus and cerebellar ataxia, which latter symptoms enabled localization in or about the cerebellum. Further, the implication of the trigeminus, sixth, seventh, eighth and of the twelfth nerves on the right side, was sufficient to localize the neoplasm more definitely. When I presented the

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patient in my Cook County Hospital clinic, I made the diagnosis of rapidly growing tumor in the right cerebello-pontile angle, and suggested immediate operation.

Operation.—Dr. Schroeder, of the Cook County Hospital staff, performed the operation on Dec. 17, 1909. A large tumor mass was discovered in the right cerebello-pontile recess, which he considered irremovable. He contented himself with making cerebellar decompression and then closed the wound.

The patient made a good recovery from the operation, but died a few days later—December 27—from the effects of facial erysipelas. Post-mortem examination was refused.

COMMENT

There could have been no difficulties in the diagnosis of a case so rich in classical symptomatology as this one. Only the unusual features therefore will be touched on.

The headache, it will be recalled, was principally in the fronto-parietal region and only occasionally in the occiput, its usual place of election. Had there been no other symptoms, this might have caused confusion in the mind of the examiner and would have created a doubt as to location. It must be recollected, however, that the situation of a headache has but limited localizing value and that in many cases of cerebellar tumor the headache has been entirely frontal.

A symptom which still constitutes a topic of lively debate among neurologists is the muscular weakness occurring in cerebellar and cerebello-pontile angle tumors, some maintaining that it is a real motor paralysis and the result of motor tract involvement, others assigning to it a purely cerebellar cause and explaining it as a sign of cerebellar asthenia. To the latter group of men belongs Luciani, who described three symptoms as characteristic of a cerebellar lesion produced experimentally, namely, atonia, asthenia and abasia. Clinicians have found this triad of symptoms in disease of the cerebellum, and particularly in tumor of that region.

In my last case a diagnosis of cerebral hemiplegia had actually been made because of a rather sudden appear-

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ance of right-sided weakness combined with facial involvement. Had the type of facial paresis been investigated, it would have been found to belong to the peripheral-nerve variety, the upper branch being implicated. The absence of pathologic reflexes in the extremities presumably paralyzed, and the uncrossed character of the asthenia—being homolateral with the several cranial-nerve lesions—negatived the diagnosis of hemiplegia and favored that of cerebellar asthenia on the same side as the tumor.

100 State Street.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. HOPPE, LANGDON AND KRAMER, AND GRINKER

DR. C. H. HUGHES, St. Louis: Papers of this character are always exceedingly interesting from the light they give us on the subject of diagnosis. Symptomatically in all we have choked disk and titubating, unstable gait. No paper has yet been given presenting a case so markedly unilateral as to give us that characteristic symptom in the gait, of a disposition to describe a semicircle in the walking of the patient.

The subject of brain tumors becomes especially interesting for the purpose of diagnosis, particularly focal diagnosis, as they impinge on the origin or a course of certain cranial nerves. Tumors of the posterior portion of the brain and cerebellum often result in loss of vision which is quite characteristic, but the tumor in the white matter of the brain causes choked disk only from the general involvement of the brain, and its congestive condition apparently, so far as my observation has gone.

When as a surgeon, I was called to preside over the destinies of an insane hospital, I used to think that it was the province of a surgeon to operate in every case when opportunity offered, especially for epilepsy and tumors. But I am not so hasty now in operative suggestion. I recall an instance of unilateral involvement of the cerebellum accompanied by the characteristic tendency to describe a circle on the opposite side of the lesion in attempting to walk; I was agreeably surprised in that case, after the use of extensive doses of cathartics and sodium bromid, etc., to see that symptom disappear. Notwithstanding, the man died and I made the autopsy which McLean Hamilton reproduced in his classical work on that subject at the time. We have not all the light that is coming to us yet on the subject of tumors, but we are getting more light than we had some years ago.

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Dr. G. W. Hall, Chicago: In one case that Dr. Grinker reports, the patient, who came on my service at the Cook County Hospital, showed the typical findings of the process in the right cerebello-pontile angle, and the question at that time so far as the diagnosis was concerned was whether or not there was an abscess or tumor. As Dr. Grinker brought out in the history of the case, it was at first very puzzling, because the patient had also a discharging ear; but the fact that the ear had been punctured by a physician, on the outside, at a more recent date than the onset of the symptoms, easily cleared up the history, sufficiently to guarantee the advisability of an operation.

In connection with that case I might mention also the conditions which Dr. Hoppe brought out as to frontal tumors. No one can make an absolutely accurate diagnosis as to localization of tumors of the brain perhaps outside of the cerebello-pontile angle. We have seen instances occur in which diagnosis of cerebellar processes have been made, and the processes were found in the frontal lobe or in the brain proper.

Dr. Hoppe's statement as to the mental symptoms occurring in the frontal tumors rather than in the cerebellar tumors seems to be a point well taken. That was especially dwelt on by Dr. Beevor in one case he had on his service in the hospital at Queen's Square, London, where he made the probable diagnosis of a frontal tumor rather than a tumor of the cerebellar process because of certain mental symptoms present in that case.

Dr. Charles L. Dana, New York: A few years ago the subject of brain tumors had become interesting, because the success of surgical intervention was so poor, but in the last four years we have been much more successful in localizing and removing tumors, especially subtentorial tumors. In historical justice to American neurologists, Dr. Grinker ought to state that the attention to the clinical facts regarding tumors of the cerebello-pontile angle, was first brought out by Dr. Hunt and Dr. Franckel about four years ago in a series of papers and reports which they made at that time; it antedated any emphatic recognition of these tumors by any foreign or American neurologist or surgeon that I know of. At that time they reported several cases and three operations, all unsuccessful; but they then drew attention to the fact of the ease with which such tumors can as a rule, be recognized. One point which they made is the fact that in some of these patients there was a neurofibromatosis of the skin, so that in some cases the possibility of a tumor of the cerebello-pontile angle was suggested by the fact that a great many little fibromas of the skin had been noticed in the patient.

There is a symptom in cerebellar disease sometimes seen in tumors, hemorrhages and softening and atrophic conditions, that is, a certain tendency to catatonia.

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The cerebellar seizures that have been referred to vary somewhat in character, but are most frequently, I think, either sudden plunges forward or sudden droppings down by the patient as though he had been struck by lightning, or suddenly fainted; at other times the patients fall down and assume the sprawling attitude and tonic spasms such as are seen in animals experimentally operated on. These cerebellar seizures generally come from lesions that begin from an extramedullary source,—tumors of the pontile angle or of meningeal origin.

Dr. Theodore Diller, Pittsburg: Those who attended the Section last year at Atlantic City will remember a patient that I showed before the Section at that time, from whom a cerebellar tumor had been removed. This patient suffered from very severe headaches, showed a high grade of optic neuritis, 6 or 7 D., and had a posture of the head to the left, away from the tumor. The tumor was removed from the anterior margin of the cerebellum after about one-third of the cerebellar lobe had been removed. I saw this patient about a month ago; he is working on a farm. His gait, which was extremely uncertain, is good. He is entirely free from pain. He says that he sees well, and is comfortable in every way, and the only thing that we can discover wrong with him is a moderate grade of optic atrophy. This case is now of about eighteen months' standing.

I commend very much Dr. Hoppe's method of presenting his case here today by the pictures. It is difficult to transport specimens to a meeting; but stereopticon views like these give as plain a perspective view of the tumor as the actual specimen would.

With regard to this point of the confusion between frontal and cerebellar lesions, let me say that recently I saw a patient whose history was that he had fallen, and that an operation had been done in the frontal region, and a small amount of pus removed a year ago. When I saw this patient he was having a great deal of pain in the occipital region; he was complaining of very severe pain confined to the frontal region and his head was extended backward very far. He had no fever; and he had not the slightest pain in the frontal region. We hurried him off to the hospital. An operation was done over the frontal region and an abscess was found immediately below the site of the old wound, and I think fully an ounce of pus came out. The case is very striking. The only thing the man complained of was very severe pain in the occipital region (not localized as to the side—probably bilateral), and this was due to an abscess in the frontal lobe.

Dr. D'Orsay Hecht, Chicago: The interest taken in cerebellar neoplasms and the discussions arising therefrom, have resulted in a gratifying percentage of accurate localizations in operations in this region. This is particularly true of cases

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with acusticus symptoms. As soon as a suspected tumor presents in a marked degree eighth nerve involvement, the tendency is to designate it as a cerebello-pontile angle tumor. I should like to get some expression from the members with regard to the propriety of calling tumors, necessarily wide-spread in their extent over the cerebellum or in the cerebello-pontile space, angle tumors. We are in truth not dealing with an angle, so far as the invasion by tumor of this area is concerned. We are rather dealing with a quadrilateral space, something that has more of the configuration of a rectangle. I feel, therefore, that if we get away from the designation of angles in these cases we may be serving cranial topography with more accuracy, so far as this site of predilection for certain tumors is concerned.

In Dr. Grinker's paper, I think that, aside from the complete report of his cases, chief interest centers in the character of his summary, in which he dwells upon the question of glioma and sarcoma, as well as the expediency of lumbar puncture. I think that we as neurologists are quite agreed that lumbar puncture of itself is a relatively innocuous adjunct to our diagnostic technic, but it may prove a decidedly dangerous measure in the presence of a brain tumor. Perhaps this particular fact is not sufficiently impressed on internists or general practitioners. I have not infrequently heard physicians who busy themselves more or less with the modern routine in neurologic examination, ask whether a lumbar puncture has been made, forgetting, I think, that lumbar puncture may be of real danger to the patient when we are dealing with obvious symptoms of intracranial pressure of tumor origin. I should advise extreme conservatism in doing lumbar puncture in this type of case.

I think, too, that in those cases in which cerebral tumor of solid formation is suspected, we are not infrequently surprised to find a cyst, capable of evacuation, and therefore yielding a better prognosis than was admissible from the premises prior to operation. I recall such an experience five or six months ago, when it was my privilege to see a case of brain tumor at the request of Dr. Alfred Murray. I made a localization of cerebellar tumor and the patient was operated on by Dr. Albert E. Halstead, of Chicago. The acusticus findings were of very material value in referring the tumor to the cerebello-pontile space (or angle, if you wish) and on a very extensive decompression, exposing both sides of the cerebellum very widely and spaciously, it was found that the suspected area was filled with a fairly large cyst, which was properly evacuated, and at a recent date, the patient was doing very well in his own home, and able to be up and about, for a time even following his occupation as a pharmacist.

I agree with Dr. Dana that we should not forsake the old and well-tried clinical criteria in arriving at a localization, but, on the other hand, I feel that we should put due emphasis on some of the newer additions to our technic and credit with importance such a symptom as, for instance, has been advanced

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by the French school, and called diadokokinesia. I have noted it in several instances, and I think, as an evidence of asynergy, it is quite as important as are other symptoms entering into the question of the coordinating function, as it depends upon the cerebellum.

Dr. Julius Grinker, Chicago: I wish to emphasize a point of general practical interest, namely, that hysteria and neurasthenia are diagnosed often in cases requiring more extended investigation. I recall several such cases of my own, in which the symptoms at first pointed to functional disease, but brain symptoms developed later and the diagnosis of organic disease had to be made.

One point which has not received due emphasis in this discussion is that cerebello-pontile angle tumor may begin with hemiplegic symptoms. My third case, which for lack of time could not be reported in full, was diagnosed as hemiplegia at a time when there were hemiplegic manifestations in the form of one-sided asthenia, that is, a unilateral weakness resulting from cerebellar atonia. In these cases there is neither exaggeration of reflexes, nor Babinski sign.

I am very thankful to Dr. Dana for correcting my short reference to the historical development of this subject. It is quite true that Drs. Hunt and Franckel were the first to call attention to tumors in the cerebello-pontile angle; they also furnished splendid contributions to the symptomatology.

In my opinion the new cerebellar sign described by Babinski under the term "adiadokokinesia" is not valueless. When present, it is a reliable sign. It is more often absent than present and frequently it only appears after an operation has been performed on the cerebellum, certainly too late to be of value in diagnosis. In one of my cases this symptom was present before any of the others. While, then, this sign may not be of the greatest value, we may nevertheless consider it, if only the observation may serve to determine its proper place in the symptomatology of cerebellar tumor.

Staggering to one side is not a reliable focal sign. In two of my cases the staggering was on the side of the tumor; in the third case the patient staggered to either side.

Concerning speech disturbance, my first case showed an utterance which might be called "jerky" speech, rather than bradyphasia. The so-called "cracked-pot" resonance I have not found in my cases, but I have often observed it in the hydrocephalus of children.

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THE TREATMENT OF ACUTE AILMENTS IN PERSONS ADDICTED TO THE HABITUAL USE OF NARCOTICS

GEORGE E. PETTEY, M.D.
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The fact that death is almost certain to be the result of an acute ailment such as typhoid fever, pneumonia or dysentery, when these ailments occur in one who is addicted to the haitual use of a narcotic drug, leads us to inquire as to the reason for this high mortality. Every internist is occasionally called on to treat acute ailments occurring in persons addicted to the use of narcotics; and in such persons these ailments present some very knotty problems which they do not present when they occur in persons not so addicted. Therefore it is proper to ask in what way and to what extent the habitual use of the narcotic modifies the acute ailment. Does it increase or decrease its severity. To what extent and in what manner will habitual use of the narcotic interfere with the effects of remedies used in the treatment of the acute ailment? How will it affect the prognosis? Shall the narcotic be continued or witheld during the treatment of the acute ailment? If continued, how? If discontinued, how is that to be done? These, as well as other equally perplexing questions confront every one who is called on to treat patients of this class when attacked by an acute ailment, and his success in such undertaking depends largely on his ability to solve these problems.

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ACTION OF MORPHIN

In an effort to find answers to these questions it is necessary to consider the effects morphin has on the system. At the very beginning of its use the first dose brings about a state of quietude or torpor accompanied by diminished excretion of the products of waste. In the course of from six to twelve hours these effects of the dose subside and the functional activity of the system becomes normal and possibly excretion is carried on at an accelerated rate, but it requires several days for this increased activity of the excreting organs to free the system from the products of waste which should have been excreted during the time these functions were restrained by the effect of the narcotic. Now, if before this is accomplished another dose be taken, the work of the eliminators is again interfered with, resulting in still further retention of the products of waste. In the drug habitué these doses are repeated from day to day, usually several times a day, thus constantly interfering with the work of the eliminating organs, and the system soon becomes surcharged with the products of tissue disintegration and their fermention compounds. In other words, the habitual condition, the normal state, as it were, of a drug habitué is one of profound toxemia. These toxins are of intestinal, drug and auto-origin.

That the source of these poisons may be more clearly understood it is well to remember that the mucous membrane of the bowel is a continuation of the skin and is therefore in a sense one of the outside coverings of the body. From this point of view any toxin or other substance absorbed from the intestinal canal comes from without the body as much so as if it were absorbed from the skin, and therefore cannot be considered as identical with poisons of internal or acute origin. Toxins of strictly internal or auto-origin are produced in the blood or tissues of the body by chemico-vital action in

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these substances. Toxins of drug origin consist of the drug or chemical itself in the system or of its oxidized products. In drug habitués toxins arising from all three of these sources are present or produced in such abundance as thoroughly to saturate every fluid and tissue of the body.

Any acute ailment occurring in one whose system is in such a toxic condition is greatly intensified, the fever, pain and all other symptoms are increased in severity, and if the attack be of an inflammatory type the inflammation is likely to partake of an erysipelattous nature and spread with great rapidity. In all such cases the prognosis is extremely grave.

TREATMENT

The treatment of such a condition is manifestly difficult because the narcotic must be continued. If the drug could be discontinued the eliminating organs could be readily brought into play and the toxic matter thrown off; but any attempt to withdraw the drug at the beginning of or during an acute ailment would precipitate a crisis which would almost certainly end in death. Therefore we are confronted with the necessity of curing an ailment, of overcoming a diseased condition, while its cause continues in active operation. Not only that, but if the patient is left to his own volition in the matter he will take his drug in increased quantities in order partially to overcome the distress incident to the acute ailment, and this would still further retard excretion.

In order to have a rational basis for the application of the therapeutic measures with which we are to combat this condition, we should reach a conclusion as to which of the excreting or secreting organs is materially interfered with and what is the extent of that interference.

The action of the kidneys is not materially affected. As large a percentage of urea and other excretory pro-

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ducts is thrown off by the kidneys of a drug habitué as in a normal person and the quantity of liquids passed is practically normal.

The action of the peptic and pancreatic glands does not seem to be materially affected. Morphin habitués can digest and assimilate as large, or even larger, quantities of nourishment under the effects of morphin as they could without it if their systems were in an equally toxic condition from any other cause. In other words, while they are anemic and their tissues are poorly nourished and digestion and assimilation are interfered with, this interference is due to the toxic condition of the system rather than to any direct effect of the morphin has on the digestive organs themselves.

The action of the liver does not seem to be materially affected. They are able to eat and digest fats in fair quantities, and bile may be found in the stools. It is not apparent that the action of either of these classes of secreting and excreting glands is affected to such a degree as to account for the extremely toxic condition present. Shall we, therefore, conclude that the remaining set, the excreting glands of the intestine are directly at fault; that their functional activity is checked to such a degree as to account for the retention of this toxic matter? I think not. Their action is regulated by the same nerve-centers which preside over the activity of the other intestinal glands. Their action could hardly be suspended to such a degree as to account for the toxic condition present without more marked derangement of the other glands having the same nerve-supply. This leaves us to place the blame on the other excretory force, the motor function of the bowel, and it is here that we find the chief trouble.

One of the earliest and most noticeable effects of morphin is an arrest of intestinal motion. In a very short time from the administration of a full dose of morphin peristalsis is completely arrested and remains absent

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for a period varying from four to twelve hours, depending on the size of the dose. In drug habitués these doses are repeated at frequent intervals and peristalsis is kept suspended during a greater part of each twenty-four hours. Peristalsis is essential to discharge of waste from the bowel. A motionless canal means a clogged canal. The eliminating organs may be likened to a sewer system with the intestinal canal as the main and the ducts of the excreting glands as laterals. If the main sewer becomes obstructed the laterals have no outlet for their product. Since they cannot discharge their waste into the main, it is retained or forced back into the system. The reabsorption of this waste from the clogged or sluggish excretion stream is the mechanism by which the systems of drug users become toxic. When this semi-paralyzed condition of the intestinal canal is overcome and active motility established in its stead, the accumulated waste is promptly discharged.

The fact that, when active peristalsis is induced and maintained, very little if any larger quantities of glandular stimulants are required to secure free movements from the bowel of a drug habitué than from one who is not using a drug, confirms me in the belief that the functional activity, *per se,* of the secreting and excreting glands is not materially reduced but that the failure of excretion arises almost exclusively from suspension of the motor function of the bowel.

STRYCHNIN

Fortunately we have one drug in our armamentarium which has sufficient power to bring about free intestinal motion notwithstanding the restraining effects of the opiate, provided it be given in sufficient dosage and at the proper time. That drug is strychnin. Ordinary medical doses, however, are not sufficient. In estimating the quantity of strychnin required in any given case, the age, weight and physique of the patient must be taken

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into consideration as well as the quantity of morphin, the paralyzing effects of which we are seeking to overcome. Young persons are more susceptible to strychnin than older ones. The short and compactly-built, in whom a fair degree of muscular tone is present, do not require as much as the tall and loose-jointed, who have flabby, atonic tissues; but the relation between the time of giving the strychnin and the morphin, the effects of which we are seeking to neutralize, is quite as important as the size of the dose.

PURGATIVES

Strychnin excites peristalsis by direct stimulation of the motor centers. Motor waves thus induced extend to all the structures which would receive them if the centers were acting normally, or without artificial stimulation. The arrest of intestinal motion by morphin is most marked during the primary effects of each dose or each succeeding dose, but as the primary effects of the drug wear away peristalsis gradually becomes reestablished. Drug-users, as a rule, take their drug only during the day or from the time of rising in the morning till bed-time, say from 8 a. m. to 10 p. m. During these waking hours the system is constantly under the primary effects of the drug and intestinal motion is very much restricted; but during the period between 10 p. m. and 8 a. m., the sleeping hours, the effects of the day's dosing wear away and peristalis becomes fairly active. It is during the latter part of this period that the excretory organs do the principal part of their work. In order to secure prompt action of purgatives advantage must be taken of this state of affairs. The remedies must also be so compounded and be given at such times as to have the acme of their effects, both as motor and secretory stimulants, occur during the latter part of this period when the system is least under the restraining influence of the opiate. To do this begin with the purgative course at 2 p. m. and give, on an empty stomach,

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a dose every two hours until 10 p. m. For an average patient taking ten grains morphin per day the following will be found effective:

℞	gm.		
Calomel	6		gr. x
Powd. ext. cascara	6		gr. x
Podophyllin	06	or	gr. i
Ipecac	06		gr. i
Atropin	0012		gr. 1/50
Strychnin nit.	015		gr. 1/4

M. et ft. caps. No. V.
Sig.—One at 2, 4, 6, 8 and 10 p. m.

It will be noticed that these capsules contain 1/20 gr. strychnin each, and that one is to be given every two hours until five such doses are given, making ¼ gr. in eight hours time. This would be excessive dosage for one not under the influence of an opiate, but it must be remembered that morphin opposes strychnin in almost all its range of action, and that we are seeking to overcome its paralyzing effects on the motor function of the bowel. Unless a quantity sufficient to do this is giver the secretory stimulants will simply stir up a storm in the upper part of the intestinal canal accompanied by nausea and other distress but the bowel will not be emptied. It is more than likely that this storm will have to be allayed by an increased dose of the opiate.

REDUCING THE NARCOTIC

During the time these capsules are being given the patient should have his usual doses of morphin, but none must be given from the time of giving the last purgative capsule until free evacuations have been obtained. The physician should take charge of the patient's drug-supply at the time of beginning the purgative course and control it from that time on.

The strychnin and atropin in these capsules will excite a fair degree of peristalsis notwithstanding the restraining effects of the opiate, and this will enable the glandular stimulants given with them to induce free evacuations from the bowel in eight to ten hours from

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the time the last purgative capsule is given, thus securing bowel movements before the time for the next or morning dose of morphin; but more certainly to accomplish this result, six hours from the time of giving the last purgative capsule, 1/20 gr. strychnin should be given hypodermatically and followed in half an hour with 2 ounces of castor oil or a full dose of Epsom salts, and both the strychnin and oil or salts repeated at intervals of two hours until the intestinal canal has been thoroughly emptied.

If the bowel begins to act before the time for the usual morning dose of opiate the patient will experience little, if any, discomfort, but if not, the opiate must be withheld until the bowel has been well emptied. This may be a stormy period, but this storm must be weathered.

If the ingredients of the purgative course and the strychnin have been properly adjusted to the case in hand the bowel will begin to act in six to eight hours from the time of giving the last purgative capsule and a number of free evacuations will have been obtained before the time for giving the morning dose of morphin. The relief afforded by this active elimination is usually such as to enable the patient to go in comfort several hours beyond the time for his dose, this he should do, thus allowing more time for elimination. When the patient has gone without his dose of opiate as long as he can without discomfort it should be given to him, but in reduced quantities. It will be found that after the bowel has been thoroughly emptied and the patient has gone without his morphin several hours beyond the usual time for his dose, not more than one-half the quantity of morphin which he had been taking will be required to meet the demands of the system. This quantity should be given at regular intervals, observing the hours at which the patient had been accustomed to taking his doses.

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Forty-eight hours from the time the first purgative course was begun, another should be started and given as the first. This course may be less or more active, according to the effects obtained from the first one. It should be followed by strychnin and saline or oil as in the first instance. This course will cause to be excreted into and carry out of the intestinal canal a residue of extremely toxic matter and the relief obtained from it is even more marked than from the first. After its action a smaller quantity of morphin will meet the demands of the system than after the first course; but whatever quantity may be found necessary to keep the patient free from abstinence-symptoms should be given; and this administration should be continued at regular intervals throughout the remainder of the acute ailment.

Following the second purgative course the bowels should be kept acting by the regular and persistant administration of both a motor and a glandular stimulant and thus prevent a recurrence of intestinal toxemia. Podophyllin, 1/40 to 1/20 gr., or sodium hyposulphite 20 gr., given at intervals of from two to four hours during the remainder of the acute ailment, will usually answer this purpose admirably, but these meet only the demand for a secretory stimulant. To insure activity of the other function essential to bowel movement, a sufficient quantity of strychnin should be given during the evening hours of each day to excite active peristalsis. With elimination thus efficiently secured and kept up during the remainder of the acute ailment, that ailment, whatever it may be, can be treated with but little more difficulty and with about as much success as if there were no drug addiction present.

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MANUAL WORK IN THE TREATMENT OF THE FUNCTIONAL NERVOUS DISEASES

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The task of treating the functional nervous diseases is so important and so perplexing that you who know the subject best will be most willing to weigh the experience of any one who has honestly investigated in the field. For this reason, although a general practitioner, making no claim to especial neurologic training, I venture to lay before you my conclusions after some years' experience with manual work as a remedy.

THE RATIONALE OF WORK

In order to understand the effectiveness of this simple therapeutic adaptation, we must assume that rational occupation of some kind is an essential of sound and successful living. We must also appreciate that in the functional nervous diseases, we shall not commonly find the patient rationally occupied but either overworking or idle. We shall also usually find that the patient, whether he is idle or overworked, has adjusted his mental and physical habits to his illness, thereby perpetuating the symptoms by making them consistent parts of his daily life. Moreover, it is in my experience almost always true that during periods of idleness or of overwork, the efforts of the patient to overcome his disease result in an accentuation and an accumulation of symptoms through self-consciousness and self-concern.

Manual work, used as a remedy, aims to introduce a new and objective interest, gradually forcing its adoption and increasing its prominence until the mental and

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physical habits of the patient are grouped about this wholesome center, rather than about the old standard of illness and complexity. It is found experimentally that this plan frequently does represent so radical a change that some at least of the troublesome nervous symptoms which may have become quite dependent on the old order drop out of sight. Manual work as a remedy, if it is appropriately and wisely used, may also, in the functional derangements, go far toward bringing about a state of self-forgetfulness, which if attained has often in itself meant a virtual cure.

SYNTHETIC VERSUS ANALYTIC METHOD

The work cure, while it has the same ends in view as the various other forms of treatment, differs radically from most of these in that its course is synthetic rather than analytic. The trend of the modern treatment of the mental element of the functional nervous diseases is distinctly analytic; the attempt is made to correct the psychoses by explaining away old fears and misunderstandings. The skilled psycho-analyst delves and probes until he has laid bare the secret mental life of the patient, thereby making the mistakes and faults of the past so evident that their correction becomes logical and so inevitable. But there are those who feel that in many cases, remarkable as are the results thus achieved, any analytic study beyond that required for a fairly clear understanding of the case may involve serious dangers, especially in the hands of the inexperienced and the unskillful. If it is true that a practical unconsciousness of self is an essential of normal living, is it not possible that the self-study necessary in these exhaustive analyses may sometimes defeat its own ends by bringing about new and unfortunate states of self-consciousness? The experienced psychotherapeutist should be able to avoid such dangers, but the average man who attempts this kind of treatment must not infrequently find himself involved in difficulties beyond

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his understanding. To those who have this fear of complications, the work cure will undoubtedly appeal, for it takes the patient as he is, and seeks first to improve the mental and moral condition by substituting for the well known confused and faulty habits of life a simpler and saner order of living.

THERAPEUTIC VALUE

If we succeed in establishing simple habits of industry within the limitations of the patient and in spite of the continuance of many untoward symptoms, the therapeutic value of such accomplishment is obvious. The neurasthenic patient who after long years of worry and over-doing has made a failure of his vocation and who has had idleness forced on him, may be by this course not only improved physically and mentally in ways that any one may see, but through the action of forces we may never analyze he becomes a man who can at last be reached by argument and who can respond fully and permanently to the stimulus of wise suggestion. I need only say that the patient, except for his natural limitations, has in this course a chance in some measure to substitute success for failure and that he may frequently go back to his old life so schooled in simple straightforward living that the vicious complications do not return in force.

The difference between the analytic and the synthetic courses is, to be sure, frequently one of sequence only, for in the end we shall have accomplished in each case a virtual analysis, and in favorable cases a logical cure. But the difference in sequence is sometimes very important, and has, I feel sure, not infrequently made the difference between order and confusion.

WORK CURE ONLY AN AID

The work cure, at its best, is, of course, but a supplement and background for careful medical diagnosis and treatment. It does not attempt to discredit any of

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the methods of treatment now in vogue but may go hand in hand with them. In certain cases it may not be appropriate at all, while again it may render all other treatment unnecessary. The principle involved in the work cure is as old as humanity but it will bear frequent emphasis. Its full recognition has, in my own case at least, made the adequate treatment of the functional nervous diseases a greatly simplified problem.

WORK CURE INSTITUTION

About six years ago, I began to consider the possibility of an institution which should make practical application of the principle of industrial therapeutics and which should serve as a laboratory for experimental research in this direction. The development of the plan, although beset with many difficulties, has been most interesting and encouraging.

Perhaps other occupations and other details would have been as well or better, but the choice fell on certain of the ancient crafts such as could be accomplished without haste and without the use and danger of complicated machinery. We have to show for our efforts a considerable industrial plant, well equipped and well situated. For many reasons, the idea has been from the first that the shops be as good as good craftsmen could make them, and that the products should command respect and interest everywhere. This end has been accomplished by making the shops essentially independent of their clinical use. At the present time we have seven workers who may be properly classed as expert craftsmen or designers. Besides these, there are several assistants who will soon assume rank as craftsmen. These people go to the shops every day and work at their various crafts. The patients are introduced from time to time as pupils or apprentices. The craftsmen who do the teaching have had enough experience with illness so that, under direction, they can successfully manage the work of the pupils. With a basis like this, the

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patient who comes as a pupil has at once the feeling of support afforded by a permanent organization. He has the example of good work always before him, and the advantage of a selling agency where his own products, as well as those of the professional craftsmen, have a chance for the final approval which comes with purchase by disinterested parties. Another advantage of a well organized shop is that it makes it possible to supply the patient with approved designs, tested materials and careful supervision, so that from the start his time and strength are not wasted but are extended in profitable channels. You will readily appreciate that these practical points are important clinically as well as commercially and artistically, for there is nothing so sure to fail as indecision and uncertainty.

The crafts which we have employed are pottery making, hand weaving, wood carving and metal working. with separate shops and separate instructors in each craft.

PRACTICAL SIDE

For three years these shops represented a very considerable expense, but during the past two years they have become nearly self-supporting, through the sale of their excellent products and quite independently of their medical use. I believe that such plants on a smaller or a larger scale may be successfully established anywhere, and I hope that the success of this venture may encourage similar experiments by men who are interested in the subject. I believe it would be a revelation to most of you to know how little argument, how few of the long and trying conversations with the neurasthenic patient are necessary even at the first, when the industrial method is pursued. It is my custom in suitable cases to explain to the patient that his symptoms are at any rate intensified by the kind of life he has been living and that we shall be able to make readjustments and corrections with greater facility when he has reduced his living to the simplest terms—to terms of

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work and rest—and when he has left behind him the hopeless entanglements of the life he has been living. The patient is very rarely asked to overcome his symptoms directly, a thing his experience assures him is impossible. He is instead given simple directions for work and rest, he is assured and his common sense tells him also, that these first easy things are quite possible and within his capacity, and that further steps may be a matter of reasonable progression.

RESULTS

In a recent article[1] I have given a list of 100 cases of functional nervous diseases treated for the most part synthetically and by the industrial method. Whether my results[2] compare favorably with similar cases treated in other ways I do not know, and it is probably impossible to judge. But I feel that in addition to a fair proportion of the usual clinical successes, the training in simple, uninvolved living has been of the utmost value to the patients.

SIGNIFICANCE OF THE PRINCIPLE

I cannot refrain from directing your attention for a moment to the possible significance of this industrial principle as applied to a field reaching far beyond the confines of the functional nervous diseases. So many people are suffering in mind and body because of the

1. Work-Cure: A Report of Five Years' Experience at an Institution Devoted to the Therapeutic Application of Manual Work. THE JOURNAL A. M. A., Jan. 1, 1910, p. 12.

2. The results of work-cure quoted from the above-mentioned article are:

Name of Disease.	Number Treated.	Improved.	Much Improved.	No Relief.
Hysteria	18	15	1	2
Insanity	5	2	2	1
Neurasthenia severe	12	7	4	1
Neurasthenia mild	20	12	8	0
Neuroses	17	8	5	4
Psychoses or fixed idea	8	1	3	4
Unclassified	20	14	4	2
Totals	100	59	27	14

In reference to the insanity cases it should be said that it is not intended to treat at Marblehead any case of insanity. These cases were accepted in the early days of the institution.

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attempt to accomplish too much, or from idleness which is not necessary, that a therapeutic readjustment would mean preventive and curative medicine on a large and important scale. Nor is achievement along these lines impracticable. Last year, in a stimulating and suggestive article, Dr. Jacoby of New York reviewed the work of two large charitable institutions in Germany[3] where appropriate work is part of the treatment of nervously broken down patients. Here and there in our own country are men awake to the possibilities of handicapped labor, both from the humanitarian and clinical point of view, and because of the really important clinical results to be secured. In the sanatoria, where such a thing has been quite unknown until within the past two or three years, productive industry is rapidly finding a place. A final and most hopeful consideration is this, that the work of the invalid and the handicapped may mercifully be kept within the limitations of the individual capacity and may still be so carefully designed and so well directed that it equals or exceeds in value much of the accepted product of those who are sound and well.

No. 69 Pleasant Street.

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3. Jacoby, G. W.: A Colony Sanatorium for the Nervous and Neurasthenic: A Much-Needed Work of Philanthropy, New York Med. Jour., April 18, 1908.

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GYMNASTICS AND MASSAGE IN THE TREATMENT OF NERVOUS DISEASES

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Perhaps the greatest field for gymnastics and massage is in the domain of nervous diseases, because the problem here is generally one of nutrition and restoration of muscular function. Every specialist in these diseases should, in my opinion, have a practical training in these branches of mechanotherapy, because otherwise he cannot intelligently direct such treatment, and a considerable portion of it should be performed with his own hands. I shall not here discuss the technic further than to call attention to a few points and procedures. Nearly all patients with nervous disease suffer from digestive troubles, constipation and poor nutrition, and in abdominal massage which should be performed by the physician, or an experienced masseur or masseuse in exceptional instances, we have, to my mind, the most valuable single remedial agent for these conditions.

ABDOMINAL AND GENERAL MASSAGE

Abdominal massage should be performed through a single covering; for instance, the undershirt, and should consist of kneading of the colon in circles by the palmar tips of one hand, with the tips of the other making the requisite pressure on the middle knuckles, this to be followed by deep stroking along the course of the colon from the ileocecal valve to the sigmoid; then by a vibra-

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tion of the entire abdomen with the flat hand applied over the navel region. Lifting of the transverse colon and stomach with vibration in Glenard's syndrome, which I might say in passing is far more common and of far less significance than is usually supposed, is at times of assistance. All other manipulations I believe to be superflous. With daily treatments until the bowels reacquire the habit of moving once in the twenty-four hours, I advise the patient to strengthen the psoas and iliacus and the external abdominal muscles by the exercise of rising from the supine position on the floor to the sitting, without bending the knees.

Whenever general massage is indicated it should be preceded, in my opinion, by passive movements of each limb, because the circulation is thereby so much enhanced that it practically doubles the value of the treatment.

The vibrator invented by Liedbeck, of Stockholm, and so much exploited in this country the past few years, has physiologically the same effect as electricity in exciting muscular contraction and thereby stimulating the circulation and metabolism, and will fail in specific remedial action just as does electricity, if blind faith instead of an exact knowledge of its physiologic effects guide its use. As a sedative to nerve irritability and pain it surpasses the electric current, but it is not nearly so powerful a stimulant to muscles and nerves and thereby to nutrition and function, but it is generally more pleasant to have applied.

The extravagant claims for massage by lay operators and ignorant practitioners, together with the confusing and repelling elaboration of technic by authors on this subject, have resulted in the avoidance of a study and use of this really valuable therapeutic aid. Many manipulations require practice, but really the art is very simple and requires mainly anatomic and physiologic knowledge.

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The principles which guide us in prescribing exercise or massage practically resolve themselves into a consideration of physiological action. Under gymnastics and massage we include also all exercise.

PRINCIPLES OF MASSAGE

We may view the human being as a machine, made up of a neuromuscular system whose activities are paramount; which is fed and oiled by the gastro-intestinal and circulatory apparatus; and we can employ the much and frequently misused term "reflex action" and say that its whole function is to convert sensation into motion. Not only is the integrity of the muscular system dependent on the intactness of ganglion cells, as shown by the rapid atrophy following, for instance, poliomyelitis; but if intrauterine disaster results in amputation of a limb, the motor cortex and spinal cells governing its activities do not develop, so that it is just as essential for the growth and maintenance of the efficiency of the machine to exercise the muscles as it is to use the neural elements.

The stimulation of mental function by exercises is strikingly shown by the success of Witmer, of Philadelphia, and by the work of the Elmira Reformatory. At the latter place in cases in which lessons could not be learned previously, the mind was virtually reached through the muscular system. I do not think that we appreciate how much the proper condition of the nervous system is dependent on the afferent impulses from the use of the muscles; it may be compared to the influence of the sensory neuron on the motor. The muscles form the furnace of the body and the greater part of our body heat comes from their use; their contractions exert a profound influence on the circulation in arteries, veins and lymph vessels, on respiration, metabolism and excretion, altogether, on nutrition.

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VARIETIES OF EXERCISE

Exercise of the muscles should mean more than the habitual use of the legs in walking about in our shops or offices, or the use of our hands in our daily vocations; it should mean some game or movements which calls into play practically all of the muscular apparatus, and at the same time pleasantly occupies the mind. To do the most good it should be a daily affair, even if of short duration, and taken out of doors whenever possible.

The physiologic action of exercise on vital functions makes it the most important single prophylactic against the development of nervous disease. Realizing that neural expenditure is greater than muscular and far more exhausting vitally in all "willed" movements, we have the key to the kind of exercise to prescribe for the individual patient. If they are extremely run down we begin with passive movements and exercises of imitation, progressing to complicated evolutions requiring cerebral initiative later. Sawing wood or any other work calling into play the greater part of the muscular system is valuable exercise. Of gymnastic systems the Swedish is easily first. Its essentials are: active and passive movements of which each joint is capable, with or without resistance on the part of the operator or patient, a proper position of the trunk, and a progressive sequence of exercises. Systems advertised by "strong men" in our newspapers, which consist of joint movements with strongly contracted muscles, develop some muscular strength but are of very little value therapeutically. Machines for exercise from the complicated and expensive Zander's to Sargent's pulley weights are very commendable. Dr. Gulick's Y. M. C. A. system of exercises is commendable. The German turnvereins make more for developing strength, especially of the upper extremities, and teach self-control, but are

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not so valuable for the rounding out of the individual as general athletics, and are not well adapted to therapeutic ends.

EXERCISE AND MASSAGE

Physiologically speaking, massage differs from exercise in that the latter has cerebral initiative, and with this, trophic impulses, and is both more intense in its general action and also more exhausting. Movements and massage increase the functional activity of the glands, of the skin, and for our purposes, massage has a pronounced anesthetic effect locally and a hypnotic effect generally. On the blood-pressure massage produces an initial rise followed by a fall, obeying in this case the same law that obtains in all forms of stimulation; namely that "reaction is equal to action;" this holds good for exercise.

FATIGUE

Fatigue is a phenomenon of double origin: one form is due to exhaustion of the nerve elements, and the other to a heaping up of work products locally in the muscles. We usually deal only with the first, as it is our consciousness of tire of the nervous apparatus governing motion that we speak of as fatigue; the impulses from the motor centers—the cerebral initiatives—exhaust the highly differentiated nerve structures first, so that a long-distance runner, for instance, after establishing the equilibrium between respiration and circulation which we call getting the "second wind," shows fatigue first from exhaustion of the nervous mechanism—from "willing" the movements—and lastly from tire of the muscles. It is probably safe to say that directing the attention to the execution of movement uses up the pabulum of the ganglion cells, while waste products accumulate, so that the fatigue of the nervous system is essentially the same as that of the muscles. Ranke washed out fatigued muscles with a saline solution, im-

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mediately restoring them to vigor, and Zabludowski restored muscles exhausted from faradization by massage. In other words removing the waste products restored working capacity.

There is another element in fatigue which is of peculiar interest to us as neurologists, and that is the mental factor. We see a young man in fine physical condition go into training for some athletic contest, living the "simple life," and then all at once begin to lose weight, perhaps developing a diarrhea or troubled sleep; in short he goes "stale"; his expenditure seems to exceed his income, nutritionally speaking. This can be explained only by the hypothesis of an exhaustion of the neural elements, of the cells governing his higher cerebration; in the last analysis, a fear in the mind. We know that emotion is far more exhausting than physical exercise. The high tension of the runner. waiting for the pistol-shot, exhausts him far more than the muscular effort of running. A neurasthenic is an individual gone stale.

NEURASTHENIA, HYSTERIA, HYPOCHONDRIA

For our purposes we may divide nervous diseases into functional and organic. The great functional diseases are neurasthenia, hysteria and hypochondria, all three of which are essentially mental troubles. Neurasthenia has been considered a fatigue neurosis, the pathology of which has been assumed to be a chronic malnutrition of the nerve cells due to some auto-intoxication or other chemical or physical disturbance. I firmly believe that in a few years we shall all recognize that the determining underlying cause is a disorder of the thought apparatus which will demand mental treatment. Breur and Freud have thrown a flood of light on hysteria and the psychology of automatisms, dreams, and various psychopathic states, and whether we treat a case of hysteria by psycho-analysis or by the reasonable persuasion of DuBois, we all recognize that the essential

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thing for a cure is to supplant the "sick idea" in the mind of the patient with the healthy one. Hypochondria undoubtedly is a disorder primarily of the mind, and when we can remove the fundamental fear we have started the patient on the high-road to recovery.

The clinical manifestations vary in these three disorders but are largely the result of perverted neuromuscular function, and as it aids in the removal of these, mechanotherapy is of the greatest utility, and besides is of value as part of the mental treatment. The Weir-Mitchell rest-cure has been a favorite measure in the treatment of these diseases, but its main value, as Du Bois says, is to give the physician mental control of his patient. There are patients who will derive benefit from this procedure, but I think that the vast majority will do better with a routine of exercise and massage graduated to the degree of functional impairment.

Gymnastics, games with balls, of which golf is easily first for the adult, horseback riding, walking, dancing, out-of-door work, rowing, fishing, and hunting are the exercises indicated. General massage combined with passive movements of the joints is of great value frequently. Since the nutrition usually is below par, abdominal massage because of its beneficial action on the "kitchen" of the economy has been of the greatest service in my experience, and will remove the chronic constipation of these sufferers if a regular habit is insisted on at the same time.

Insomnia is a symptom which is again a disorder of thought mainly, and removal of this is the important thing, but an important aid is general exercise and the general massage treatment; especially when succeeded by sedative peripheral stroking with the hand or a brush, administered at bedtime.

If we stop to think of it, nearly all our so-called functional nervous troubles are essentially "thought" disorders, and exercise, gymnastics and massage are valuable aids to the mental treatment.

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MASSAGE IN ORGANIC TROUBLES

Organic troubles can all derive benefit from movements and massage. In some diseases, such as the spastic spinal disorders, care must be taken to be very gentle in all manipulations or the treatments may easily do as much harm as good. The paralyses following acute anterior poliomyelitis appear at first glance hopeless, but in no other serious impairment of the use of the muscles can such brilliant results be obtained as here. Massage, active and passive movements with and without resistance, and the maintenance of a correct position of the limbs by suitable orthopedic apparatus will restore life and function to withered and apparently dead members. To my mind these results must depend partly on a central stimulation of ganglion cells from the periphery, aided by impulses coming from the cortex. We should stimulate the latter by encouraging the patient to "will" the movements which we execute passively, before any movement is possible by the patient.

Locomotor ataxia demonstrates well what possibilities lie in persistent practice of movements. Frenkel accepts the anesthetic theory of ataxia, and believes that the persistent practice of the exercises bearing his name compensates for the absence of the sensation of the movements; while Goldscheider thinks that through these exercises, the irritability of certain uninjured cord neurons is heightened and in that way act vicariously for the degenerated pathways. These exercises depend on the degree of disability and are not difficult of application; some are to be made in bed, others sitting, standing or walking. Fatigue is to be guarded against, and the movements must be executed with methodic precision. In my own experience the practice of the elementary movements of walking are of the greatest utility to the patient. Massage and movements are of the greatest aid in keeping up nutrition and function in the imperfectly used muscles and joints.

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Facial palsy responds kindly to massage in conjunction with galvanism and other measures.

The various forms of occupation neuroses such as writers cramp should be treated by coordinated muscular exercises which do not call into play the same group involved in the cramp, and with these passive movements and massage are to be used.

In the various forms of neuritis, when inflammation has subsided, as indicated by the absence of pain, movements and massage are of the greatest value.

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ABSTRACT OF DISCUSSION

Dr. C. R. Woodson, St. Joseph, Mo.: Employment of a congenial nature, either in or outdoors, is beneficial in the treatment of some nervous troubles, and is not necessarily confined to functional forms. Employment outdoors ordinarily brings better results than indoor employment, from the fact that there is more diversion in it. And outdoor employment brings a little fatigue and necessarily has associated with it some sunshine; fatigue and sunshine have much to do in bringing appetite; and the three combined are Nature's best remedies in producing sleep, and that is the greatest nerve restorer that we have. There are many persons who suffer from nervous diseases who do not obtain relief of the pent-up forces without employment, and it is especially good for an exalted type of mental disease, particularly where it has passed the exhaustive stage or period. Take the commonest cur and tie it up and it becomes vicious as a bulldog; but let it give off the pent-up forces and it becomes so docile that our little children can play with it.

In regard to massage and gymnastics in the treatment of functional nervous diseases, in some respects I agree with the essayists, but I have found that massage is more commonly beneficial with organic disease. Massage is good where there is defective circulation; in the old it is good in the various paralyses; especially is it good in the treatment of poliomyelitis; that is, associated with other things, and gymnastics; or I will even stop short of gymnastics and say, exercise. The fact that you teach the child that suffers from poliomyelitis to try to do the things that it cannot do, is beneficial. Employment in the treatment of those who are congenitally defective, or those who suffer from acquired defectiveness, may be beneficial because of the close relationship of the hand and mental centers. The individual who suffers from nervous troubles of a functional nature, if young, in many instances may be benefited by gymnastics, but they are not appropriate in

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the old. That massage does help to correct many defective metabolic changes there can be no question, and it should be used; but to lay down a rule of treatment, employment in industrial buildings or outdoors, or massage and gymnastics, is as broad as taking any one of the numerous therapeutic agents and saying it is good. They are all good, but it requires much more than these to effect a cure in most cases. In melancholia in which the individual's mind is not impaired, but dwells on his miserable condition, employment takes the patient's mind off from himself, his appetite increases and sleep comes, all helping to effect a cure.

Dr. John Punton, Kansas City, Mo.: Of interest to the neurologist as well as to the internist is the care and treatment of the neurasthenic, especially the poor neurasthenic, the man or woman without means such as are found in all large cities. I think that Dr. Hall deserves credit in suggesting to us the method which seems to me to practically solve that problem in many cases. When he can institute and organize a method of treatment which after a few years of experimentation has become so practical in all its methods that it becomes self-supporting, it does seem to me that our state authorities can well afford to introduce in the larger cities at least a psychopathic hospital where many of these poor unfortunates can be treated with just such treatment as he suggests, and in a very short time could be made self-supporting, so that the state itself would be at no great expense.

Dr. Hall did not state in his paper whether he used other treatment or not. I presume that he did; but if he did not I cannot quite understand how he can meet all cases. Those having some experience with the treatment of this class of patients which he referred to, know as well perhaps as I, that many of them are wholly exhausted when we first see them; in other words, I believe that there are degrees of exhaustion even in neurasthenia, and many times it depends on the state that you find your patient in as to whether or not this method of treatment would apply at the time that you see him. But I believe that there comes a period in the course of the disease when this method can be put to good use in most cases.

It was especially interesting to know that Dr. Hall recognized the fact that it does not apply to all cases of neurasthenia. I believe he is correct, because I can well understand why some of the neurasthenics could not be satisfied with this method of treatment; but the large majority of them could be greatly benefited by it, and I think that the doctor deserves congratulation on his success.

Dr. Frank R. Fry, St. Louis: I want to commend Dr. Hall's careful methods. They are by all odds the most important contribution to employment therapy that we have had

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in this country. In the course of time he will receive due credit.

There are many of us—all of us, in fact—who practice neurology, who know that a great deal can be accomplished by work-therapy. In this instance, as in other similar matters of therapy, we do not get at it right. For instance, we are too apt to send the patient to somebody to get massaged, and too often that is about as far as we go with it. We should know the details of massage better. The same is true of exercise. I do not believe that anybody ought to attempt to prescribe exercise for a patient in a case of any importance, unless he is familiar with some of the processes of gaining exercise; unless he knows something technically about what exercise means. For instance, a patient cannot walk because he has bad feet; he must have exercise; another one because the pelvic contents are uncomfortable when she attempts to walk must have other exercise. How are we going to prescribe exercise unless we are fairly familiar with the methods of getting it? This work that Dr. Hall and Dr. Sneve explain in their papers so well is important. Gradually a larger contingent will gain the kind of intimacy that is necessary to make it effectual. We must recognize the rapidly increasing importance of all of these methods, and the necessity of availing ourselves of them if we are going to hold our own in America with neurologists in other countries.

Dr. Philip Zenner, Cincinnati: I wish that Dr. Hall would manage to read a paper of that kind often, and try to interest not only medical men, but the public, in it, so that we possibly might get what Dr. Punton spoke of, some assistance from the public or from the state for these purposes.

There is one thing the doctor mentioned in connection with occupation treatment that is important, and that is, that it often takes away from the patient's mind the effort to control himself, the effort that he makes over himself to overcome his symptoms and to get well. That is often a difficult point with us. Effort means everything to the patient in order to get well; and yet we see many cases in practice in which that effort doubtless is one thing that is keeping him from getting well. It is difficult sometimes for us to decide what to do in this matter, and very often the wisest thing is to tell the patient not to make any effort over himself.

Dr. C. P. Emerson, Clifton Springs, N. Y.: A visit to Dr. Hall's sanatorium interested me so much that I have tried this therapy in the many cases that come under my notice each year. It is true that among a certain number occupation therapy is valuable. For instance, the carpenter-shop is a very good thing for many clergymen and school-teachers, but not among the farmers or day laborers.

I was much interested while connected with the Johns Hopkins Hospital, and working in the social charity work of the

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city, to find how many society persons were willing to do in the way of occupation therapy, social work among the poor sick. Of course our purpose is to decentralize the person, but nevertheless it seems to us that what is needed first is a good, thorough rest, a good course of purgation, a very, very simple diet, hours and hours out in the sunlight and fresh air, without any physical exercise. Then the occupation therapy is in order. One must study the case. What work will interest and decenter the person? It may be this, that, or the other; art for some, the carpenter-shop for the clergyman and school-teacher, who have spent most of their lives in closed rooms; and the corn field, let me say, after the patient is well rested, is also better for him than a Zander apparatus.

Dr. L. H. Mettler, Chicago: This question of exercise in the treatment of nervous diseases is a much broader one than would appear in the discussion of it in these papers. The last speaker, as well as Dr. Punton, has intimated that which seems to me to be the most significant point in the whole matter, namely, the establishment of the particular character and purpose of the exercise to be employed and the particular disease in which the exercise seems to be called for.

Many perfectly healthy individuals would derive benefit from the discipline and activity involved in the manual training of the workshop, the systematic reading of select literature, or any other means of physical and mental activity. We have all seen plenty of people, apparently in a state of perfect health, to whom gymnastics might be of the greatest advantage. To iterate and reiterate that exercise is a good thing in the treatment of nervous affections, without further specification of the why and wherefore, is a useless waste of time. Indeed it brings to mind the old story of the physician who advised his neurasthenic patient to take more physical exercise, and found his advice to be a bit ridiculous when the patient slyly informed him that he was a letter carrier. The trouble with all such advice and with papers like the second one that we have just listened to, is that they are so beautifully indefinite. The grain of truth discoverable in the midst of all their elaborate phraseology is that walking, bathing, gymnastics and exercise are highly commendable because they somehow improve the circulation. Such an ancient saw, however, does not call for any elaborate expenditure of ink and paper in its exposition. It would be far more to the point, especially before such an audience as this, to give some detailed description of the exercise, the nature of the disease in which the exercise is to be employed, and the therapeutic relationship between the selected exercise and the pathological process in question. As Dr. Fry intimated, such a discussion of gymnastics as we have just listened to, makes one think of the exploitation of certain serums. It amounts to saying, "It is a good thing; give it!"

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And again, let me say I am amazed that anyone, like the reader of the second paper, should have the hardihood to rise up before a neurological audience, such as this, and with a wave of the hand dismiss hypochondriasis, neurasthenia, psychasthenia and hysteria as being all more or less alike in representing merely some form of mental trouble. Many neurologists, I know, would be most happy if they could settle so satisfactorily the many difficult problems connected with these distinct and long recognized clinical entities. I am still of the opinion that many of the neurasthenic cases that I see are far from being merely psychasthenic. In such cases I have been able to detect weaknesses and deficiencies that represented something more than merely a psychic disturbance. They may have been denominated functional or incipiently organic, but they certainly occurred in the physiologic rather than in the psychologic sphere. In their treatment absolute rest, exalted nutrition, and the overcoming of intoxication of some sort have effected cures where psychotherapy would have utterly failed. In some of these cases exercise and gymnastics would have proved disastrous. And only after proper eliminative, restful, and highly nutritional treatment, when the patient was well along toward convalescence, would selected exercise, both physical and mental, be recommended. I desire most emphatically to assert that the indiscriminate recommendation of exercise and gymnastics as a grand and useful therapeutic measure, without such recommendation being always and everywhere guided and guarded by a full realization of the nature of the disease process for which the exercise is being commended, is close to the border-line of quackery and is wholly unwarranted in the light of modern neurology. It will be the cause of much damage and will be recognized by every intelligent neurologist as a form of malpractice.

Dr. Theodore Diller, Pittsburg: In the employment of any therapeutic agent we must use discrimination; and I assume that the gentlemen who have advocated work would of course apply these therapeutic measures with discrimination; the whole treatment of functional nervous diseases therefore comes down largely to a matter of personal equation. Some one has said that no one should become nervous until he becomes rich. We know how badly we are handicapped in the treatment of nervous diseases by lack of financial resources. I recall that Lord Salisbury some years ago made a very able address, which I have often recalled, in which he made the statement that the greatest single advantage—surpassing all others—of surplus wealth, was the confidence and resources that it gave us in case of illness. With wealth one can rest without worrying about financial matters; he can procure the best doctors and nurses, and he can travel or take a long vacation.

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Now with this generalization I should like to express my warm conviction that there is a therapeutic agent which goes with massage, with exercise and with rest, and which I believe is extremely important in the treatment of functional nervous diseases; I mean having fun. I have long been convinced that all other things being equal, the very best way to get exercise is by a game while having fun. We know that this is true of healthy children; the best way that they get exercise is by a game. Contrast the boy whitewashing the fence with the boy playing baseball. We have golf coming along now, and I should like to see it put in all our parks. I believe in this spirit of fun, and the more you can make exercise a game, the better it will be. Of course these other things have their peculiar and especial application, and I think no one can doubt the value of regular work for many nervous persons. I believe that it is one of the greatest things, not only for patients with functional nervous diseases, but as a promoter and conserver of good health.

Dr. Herbert J. Hall, Marblehead, Mass.: I tried to make it clear that I have not confined myself at all to industrial treatment. I stated in my paper that the work cure at the best is but a supplement and background for careful medical diagnosis and treatment.

Dr. Haldor Sneve, St. Paul: I wish to commend Dr. Hall for the valuable new procedure that he has described to us. But I want to utter a word of warning that we should not treat it as a routine cure-all, and prescribe the latest fad. We are prone to faddism in medicine, and especially in neural troubles I believe; and I want to say that I think the main element of cure in Dr. Hall's method, if it may be called that, is the mental element. I am sure Dr. Hall recognizes that, and I want to utter a word of warning that it is not just because you do the work and occupy yourself that suffices, because if we did not recognize the mental element behind it, the treatment would fail.

Now as to general gymnastics, exercise, massage, etc. I took particular pains in the few observations I made in this paper to say that these are not to be prescribed indiscriminately, and the paper really resolves itself into a consideration of the physiologic action of gymnastics and massage; and to lay down, as one of the last speakers said, definite rules, is impossible. You must use the gray matter in your brains. You must understand the physiologic process involved. We cannot lay down primer rules in things of this kind; and they must remain more or less indefinite.

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NERVOUS AND MENTAL DISTURBANCES OF THE MALE CLIMACTERIC

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It may be acknowledged at once that the term "male climacteric" is a bit sensational, in view of the fact that it is not specifically recognized by any physiologist. For instance, the "American Text-Book of Physiology" denies that the male presents either a monthly molimen or a climacteric comparable to that in the female. If we confine our attention on the subject of menstruation entirely to the monthly flow, and see in this rhythmic manifestation nothing but an ovarian activity, we need go no further in the discussion of the question of a male climacteric.

But it is a well-established anatomic fact that the early months of embryonic life show no sexual differentiation, and that all of the constituents that subsequently under variation develop into the sexual apparatus of male and female, are indifferently present in either sex, not only up to a certain period of intra-uterine life but in a rudimentary form throughout the entire period of human existence. The statement is a common one that before the age of puberty boys and girls are physically much alike, and it is also a matter of common observation that old men and old women again become alike both mentally and physically through the loss of sexual differences.

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MONTHLY RHYTHM IN MEN

We must, however, take a broader view of the monthly periodicity which shows itself so significantly in the female. This matter has been carefully studied, among others, by Campbell.[1] He lays stress on the fact that the monthly rhythm affects females not only before the appearance of the menstrual flow, but in the post-menstrual period for an indefinite time that extends in some instances to the end of life. He believes, although he brings nothing of great significance forward to prove it, that the male is similarly the subject of a twenty-eight to thirty-day rhythm, and his conclusions are that man, like woman, passes through monthly cycles, which begin with the beginning of life and continue until death, and that menstruation as ordinarily understood, that is, the monthly flux, is a reproductive function which has, in the case of the human female become engrafted accidentally, so to speak on this primordial rhythm.[2]

Bearing on the subject of the monthly molimen, a number of studies have been made. A German observer carefully followed the temperature in a number of soldiers living under uniform conditions, and found that, as in the case of women there was a distinct monthly variation or curve. Unfortunately, the name of this observer and the reference have escaped me.

Dr. Clelia D. Mosher[3] as a part of a long study of the arterial pressure conditions in women, with particular reference to the menstrual period, followed the same plan with five men. She found in nine women and five men thus observed for a long period of months, that there was a rhythmic fall of blood-pressure occurring in both at definite intervals. The fall in pressure in women occurs near or at the menstrual period, preceded by a preliminary rise three to five days before the

1. Campbell, Harry: Differences in the Nervous Organization of Man and Woman, London, 1891.
2. Campbell: Differences in the Nervous Organization of Man and Woman, p. 274.
3. Bull. Johns Hopkins Hosp., April, May, June, 1901.

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onset of the main fall, and these variations are not peculiar to either sex in the general features of the pressure curve or the periods of high and low tension. Both men and women who have a tendency to digestive disturbance find themselves likely to be more subject to such disturbance at the period of low pressure; or when a slight chronic catarrh exists there may be marked increase of symptoms at that time. With the high pressure, which in women precedes the flow for some days, in both sexes there is a sense of well-being, of superior activity in all directions, and apparently an increase in the resistive powers of the body against fatigue and infections.

Dr. W. T. Belfield of Chicago advises me in a personal communication that he has followed the variations in weight and temperature in two healthy young men for a period of three months, and that he found a distinct tendency, which he would not denominate a wave, toward a rise of weight but not of temperature, about every two weeks, with a phosphaturia analogous to that presented by women just before menstruation.

Sanctorius (1728), the great Italian clinician, was perhaps the first to suggest a monthly cycle in the organism of men. He based the suggestion on a careful study of the weight of the body with reference to the amount of excretions, and found that a monthly increase to the amount of one or two pounds occurred in men, followed by a critical discharge of urine, the crisis being preceded by feelings of heaviness and lassitude.

So, too, Stevenson has insisted that menstruation is not confined to the female sex, and that there also exists in men a cyclic wave which is really a manifestation of a general law of vital energy.

A number of observers have attempted to establish a cycle of sexual impulses in men, but for obvious reasons, though their findings are somewhat suggestive of such being the case, they are not sufficiently definite to justify any generalizations.

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Julius Nelson, Stanley Hall, Perry-Coste and other observers have been able to note a monthly rhythm in the frequency of nocturnal emissions in men living a chaste life.

A number of morbid conditions also seem to present such a monthly regularity as seems to be significant in this connection. I have the histories of two carefully observed male cases of epilepsy, extending over five and seven years, respectively, in which the attacks have occurred with as much regularity as the menstrual function in women, at periods of from twenty-eight to thirty-four days. This rhythm in each instance was occasionally broken by the use of large doses of bromid at the time of the expected attack, but again resumed its regularity on the withdrawal of the bromids. In both these cases the onset of the disease was at the age of puberty. Neither have attained an age that would present any modification due to involutional processes.

I have observed a case of migraine in which the attacks in a male varied from twenty-four to thirty-six days, over a period of several years; and the patient asserted that these attacks had been of similar periodicity for a number of years before he came under my notice. Incidentally, I have repeatedly seen migraine in the male subside between 55 and 65 years, just as it does in women at the climacteric, and typical periodical drinking in men usually follows the same rule.

Campbell[4] reports an interesting case of a man 62 years of age, who for twenty or thirty years had suffered a monthly hemorrhoidal flux; so regularly did it occur that he spoke of these periods as his "monthlies." Far from feeling any evil effects from the bleeding, he invariably experienced great benefit, the improvement expressing itself in improved vigor and more perfect bodily health. At the age of 60 the flux ceased to appear regularly and then he began to experience a long

4. Campbell, Harry: Flushing and Morbid Blushing, London, 1890, p. 110.

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train of minor nervous symptoms, such as appear in the climacteric in women. He suffered from constant flushes of heat, during which he felt "groggy." These heats ascended and were attended by palpitation. He also suffered from marked tenderness of the scalp, and became highly emotional and almost hysterical. All these symptoms were greatly relieved by the sudden recurrence of a profuse hemorrhage. After this occasion he did not lose any blood for two or three months, and then it reappeared irregularly for eighteen months. All this time he was liable to the same nervous symptoms, but finally the flux resumed its former regularity, after which time he considered himself in perfect health.

Somewhat similar cases are also to be encountered here and there in the literature, as, for instance, one recited by Brierre de Boismont.[5]

Gall noted in a man a hemorrhoidal flux of a periodical character similar to the case already detailed.

Chopart cites a curious case of a soldier who had a monthly discharge of bloody urine; and a similar case is reported by Rayer.

Havelock Ellis,[6] who has given this subject as much attention as anyone, reports with great detail a case of cardiac asthma, the attacks of which recurred at about thirty-day intervals for many years.

Sir Henry Halford[7] as long ago as 1831, described a condition which he termed the "climacteric disease" occurring in men between 50 and 75 years of age, but did not make a close analogy between this condition and the climacteric of women.

The involutional period of male life has frequently been referred to as a climacterium in a rather loose manner, the writers not apparently intending to imply that there was a monthly variation in the male. For

5. De la Menstruation, p. 136.
6. Ellis, Havelock: Studies in the Psychology of Sex, F. A. Davis Company, 1900.
7. Essays and Orations, London, 1831.

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instance, Freud[8] says very definitely: "There are men who show a climacterium like women, and merge into an anxiety neurosis at the time when their potency diminishes."

MANIFESTATIONS OF MALE CLIMACTERIC

If, as seems not altogether improbable, the human male shows a monthly periodicity, there can be little surprise in observing manifestations of a similar character at the involutional or climacteric period in both sexes. Since my attention has been directed along this line—that is, for the past ten years—I have accumulated a large number of observations on which I purpose to outline certain generalizations, without burdening this communication with the details of case histories.

Involutional insanity in men, as in women, is of tolerably frequent occurrence and presents practically no distinctions and no regular form of psychosis. I find that in both sexes there is almost invariably a well defined tendency to mental instability, frequently well-marked family histories of insanity or of the major neuroses, and often former attacks of mental alienation in the patient himself. The particular interest of my subject does not pertain to the insanities, but to minor psychoses and neurotic disturbances. These, one and all, however, have a mental background.

Aside from the insanities above alluded to, any considerable degree of elation, delirium, or maniacal manifestations finds no mention in my case records. Almost invariably, there are expressions showing an anxious tone of mental feeling. This varies from the actual anxiety neurosis of the Germans, with all the obsessions and phobias that mark the psychasthenic, to the rather simple anxieties of the neurasthenic and the hypochondriac. Naturally this mental attitude is attended by more or less depression, and if the patients have had an attack of insanity earlier in life or come of a tainted

8. Selected Papers on Hysteria, p. 143.

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family, fears of mental overthrow bulk largely in their list of worries. I have been struck with the frequency of various adolescent forms of mental disturbance among these early patients.

The mental features of worry and anxiety tend markedly to fluctuate, but I have been unable to notice that the fluctuations show any monthly periodicity. Daily alternation is much more marked than in similar neuroses earlier in life, and very commonly such patients will be tolerably bright and free from their worries and anxieties in the latter part of the day, the early waking and morning hours being those of decided depression.

On the physical side almost invariably there is a loss of weight and the patients present a higher arterial tension than has seemed to me to be altogether attributable to their age or general physical condition. As they tend to improve the arterial tension subsides to some degree. Gastro-intestinal activities are reduced. Indigestions and all sorts of intestinal complaints are common. These patients show easy fatigue and some general neurasthenia.

Almost invariably the patients who have been subject to periodical neuroses, such as migraine or periodical drinking, lose these attributes. As a rule the men shun alcohol, are willing to give up tobacco, and readily modify their habits in any direction advised. I have notes of one or two men who had histories of periodical drinking, who became for a period of several years steady drinkers; that is, they began to take two or three drinks a day, a practice which had been foreign to their entire life-history and was finally followed by complete abstinence. In another patient steady excessive drinking developed.

Headaches, oppressed feelings in the chest, sudden sensations of an alarming nature, especially vertigos, are commonly described. Cold extremities and heat in the head are generally mentioned.

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These conditions run a variable course of from eighteen months to three years, after which the patients regain a fair degree of their former mental and physical characteristics and go along comfortably, with naturally some reduction of their former capacities.

MANAGEMENT

The management of these patients is a task requiring extreme tact, great patience, and a variety of expedients. For some, change and travel answer fairly well, although they are not happy in it. A reduction of the daily work is almost imperative in every case. General measures which serve to ease and improve the general circulation and build up the physical health, answer a good purpose. Vasodilators have seemed to me to be of essential value. Any tendency to sclerotic processes in arteries, liver or kidneys—conditions, of course, commonly found at the age presented by patients of this class—have their own indications. On the whole, the development of any of these nervous and mental manifestations in men between the ages of 50 and 65 is not of itself of serious import.

I have felt that the recognition of these climacteric features would more frequently enable physicians to take a hopeful view, and thereby maintain a more encouraging attitude towards this class of sufferers.

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AMYOTONIA CONGENITA

THE REPORT OF A CASE WITH MUSCLE BIOPSY

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There are two excuses for presenting a paper on amyotonia congenita before this important scientific assemblage: first, the fact that this contains the earliest reported case in the United States in which a biopsy had been performed and a microscopic examination made of the excised muscle; and, second, the paucity of cases that have appeared in the American literature. There is very little material on this subject in the text-books; and the statement holds true for special works on neurology as well as pediatrics.

As yet we can not give the malady a distinctive pathologic description, one which will distinguish it from the myopathies, although I do believe that it is entitled to be classed as a distinct disease. It is a rare neurological condition, but has no doubt presented itself in the past to clinicians, to be placed in the category of one of the muscular dystrophies, polyneuritis or some other disease with palsies.

HISTORY

H. Oppenheim[1] in Berlin was the first to call attention to this group of cases in 1900. He had seen up to that time several patients whose symptoms were noticed first during the earliest few months of life up to the

1. Oppenheim: Ueber allgemeine und lokalisierte Atonie der Musculatur (Myatonie) in frühen Kindesalter, Monatschr. f. Psychiat. u. Neurol., 1900, viii, 232.

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second year, and he could find nothing in the literature covering this complex of symptoms. The diagnostic symptoms given by Oppenheim are hypotonia or atonia, much diminution of active movements, to an almost complete palsy, especially of the extremities, more frequently involving the legs in a greater degree, with no or little apparent loss of volume, a peculiar relaxation and soft feel of the muscles on palpation, marked decrease or abolition of the tendon-reflexes, a decided quantitative reduction of electrical excitability but never a reaction of degeneration. Sphincters, sensation, special senses and intelligence never showed any disturbance. The disease is confined to the skeletal muscles. The diaphragm was not involved. It appears to be a congenital trouble though in some instances it is not observed until many months after birth.

Oppenheim[2] in 1904 again reports briefly the history of a new case designated this time as "Myotonie congenita." Since that time several cases have been reported by his pupils[3] and other German workers.

The Italian literature[4] contains some early reports of cases. Some valuable additions have been made as a result of case reports and investigative work by French neurologists and pediatrists. The contributions from the English scientists are noteworthy. Especially may be mentioned the cases reported by J. S. Collier.[5]

The earliest case reported from America was by Spiller,[6] who had the opportunity of performing the first autopsy. I have reviewed closely the case reported by Orbison[7] in 1909, which was classed by him as amyotonia congenita, but which more probably was

2. Oppenheim: Ueber ein Fall Myatonie congenita, Berl. klin. Wchnschr., 1904, p. 255.

3. Vierordt: Deutsch. Ztschr. f. Nervenh., 1900, xviii; Kundt: Thesis, Leipsic, 1905; Rosenberg: Deutsch. Ztschr. f. Nervenh., 1906, p. 130; Heberman: Am. Jour. Med. Sc., March, 1910.

4. Muggi: Pediatria, 1903, p. 179; Berti: Contribuzione alla atonia muscolare congenita di Oppenheim, read before Soc. ital. di pediat., Bologna, 1905.

5. Collier: Proc. Roy. Soc. Med., 1907-1908, i, Neurol Sec., p. 60; Collier and Wilson: Brain, May, 1908; see also footnotes 9 and 10.

6. Spiller: Univ. Pennsylvania Med. Bull., January, 1905.

7. Orbison: Jour. Nerv. and Ment. Dis., April, 1909.

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some organic disease of the central nervous system which has not been excluded in a differential diagnosis. A diagnostic lumbar puncture and a serum test for syphilis should have been made. During the writing of this paper Griffith's[8] case seen in Philadelphia has come to my knowledge.

Collier[9] has collected, all told, forty cases from the literature. More recently Griffith finds forty-eight. I have been able to find forty-four including my own. There are a few cases reported in which there is some doubt about the diagnosis. I am not so sure but that duplicate reports of a very few cases have gone on record.

NOMENCLATURE

The nomenclature is already overburdened with names though our knowledge of the existence of the disease covers a period of only ten years. The first description appears under the title "General and Localized Atony of the Musculature (Myatonia Congenita) in Early Childhood[1]." Oppenheim's[2] second report comes out under the title of "Myatonia Congenita." The name "amyotonia congenita" first appears in the literature in reports by Collier.[10] A few writers, especially Italians, have used the name "Oppenheim's disease." "Congenital muscular atony," "congenital hypotonia" and "congenital amyoplasia" are other titles that have been employed. Myotonia congenita, or Thomsen's disease, may be confused with myatonia or amyotonia congenita on account of a marked similarity in names, but the two are widely different neurologic diseases. A recent text-book on nervous diseases has confused the two terms in its index.

I favor employing "amyotonia congenita," which is concise and a good clinical representative. More discoveries regarding the pathology and exact etiology may or may not indicate changing the name.

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8. Griffith, J. P. C.: Amyotonia Congenita, THE JOURNAL A. M. A., May 21, 1910, p. 1712.
9. Collier and Holmes: Brain, November, 1909.
10. Collier: Brain, 1907, xxx, 146.

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CASE REPORT

The real incentive for presenting this paper to-day is a consultation case that came under my observation recently, a report of which is as follows:

Patient.—Ethel S., female, aged 22 months, was taken on Jan. 6, 1910, to my office by the parents, who reside in the interior of Kansas.

Family History.—The father, whose occupation is that of a carpenter, presents a healthy appearance. He denies any chronic illness, including syphilis. The mother is a plethoric woman and has had no protracted illness. She asserts that she had good health during the intra-uterine life of the patient. She had only one previous pregnancy, which ended in abortion at the end of the second month. Any bad heredity which might have a bearing on the case is denied. The maternal grandmother died from the third apoplectic stroke at the age of 60.

Personal History.—The patient was a nine-months baby and had a normal delivery. The child was nursed by the mother until twenty months old. The feeding of additional food was begun in the sixth month. Teething began at the tenth month. The baby never had any convulsions, could support the head and maintain a sitting posture when six months old. She has never crawled or attempted to walk; began talking at nine months by saying mamma and papa. The child has never cried very much. Control of urination and defecation was acquired at the twelfth month. She has been exceptionally free from acute illnesses, the only one recalled by the parents being an attack of diarrhea, which was mild and ceased in five days. The parents did not perceive the child's malady until the fourteenth month, its inability to use the legs and walk giving the alarm. They consulted a doctor when she was fifteen months old and learned that all the extremities and trunk were involved in the motor weakness. There has never been any tremors. She was treated with 5 grs. of potassium iodid over a period of three months before being seen by me. Electrical treatment had been furnished by a small family faradic battery.

Examination.—The child, when held by a parent or seated, gives a first picture which does not indicate any apparent illness or defect. Nutrition is good. The extremities appear to carry about the normal volume of soft tissues for one of her age. No deformities of the joints or bones are seen. The skin has a healthy, ruddy appearance. There is not the least indication of rickets. The mental development is up to the

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normal standard. She is attentive, observing and takes an interest in all that is going on about her. Speech and vocabulary are now well developed for her age, and enunciation quite distinct. She has little fear of strangers, is free with her smiles and does not cry readily. The child is attractive, seems unusually good-natured and would excite no suspicion of any retardation of the development of her cerebral functions. Testing each cranial nerve as well as could be done in a child so young, brought forth no evidence of disturbed function, except in the seventh, whose dependent muscles seem to have a trivial weakness, this being particularly observable in an impaired power of expression during changes of mood. All the extrinsic and intrinsic ocular muscles are normal. Hypotonia, one of the fundamental symptoms on which to base a diagnosis of amyotonia congenita, is present in a decided degree in this patient. All the articulations of the upper and lower extremities have excessive joint movements to passive manipulations. Each foot can be placed with ease behind the head and neck. The arms and legs hang flaccid. The mobility of the spinal column in all directions is exaggerated. Motor weakness is evident to a marked degree for all movements of the extremities, the difference between the upper and lower being not great, but possibly slightly greater in the latter. The trunk and neck muscles are comparatively stronger. She experiences no difficulty in holding the head erect or maintaining a straight spine when seated. The muscles for the finer movements of the toes and feet, and especially the fingers and hands, are less affected than the grosser, more proximal groups. The muscles feel soft, flaccid, and there is no appreciable difference between the active and resting states. The atrophy is not apparent to sight on account of the fatty replacement and a generous layer of subcutaneous adipose tissue. The range for all movements remains unimpaired. The weakness of the deltoids is especially marked. Walking is impossible. When nearly all the body weight is supported by a second person, steps can be taken, though with some difficulty. There is no disturbance of coördination. All the deep tendon-reflexes are abolished, including the patellars, Achilles, supinators, biceps and triceps. The epigastric and abdominals are absent. The conjunctival, corneal and pharyngeal are present. There is no plantar response, tested by the Babinski and Oppenheim methods. Many of the muscles failed to respond to a strong electrical current; others of the extremities respond feebly to a strong faradic and galvanic current. None gives the reaction of degeneration. No disturbance of the various forms of sensation is found. All the special senses seem keen. No subjective pains or discomfort to manipulations are found.

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Treatment and Course of Disease.—On May 11, 1910, a report was received from Dr. Shonkwiler, the family physician of Burns, Kansas, to whom, after the consultation, the patient was returned for care and treatment. He states that she seemed to be losing ground for a while after returning home. The helplessness increased as she gained in weight. In February a light attack of pneumonia was contracted, from which a good recovery was made in three weeks. The mother noticed that following this acute illness the baby was able to handle herself much better. The present status is one of considerable improvement in motor power. The mental development continues good. The arsenical treatment, which was not well tolerated, was discontinued early. Regular massage has been given. Strychnin in small doses has been employed. There has been some dieting to prevent too much increase in adipose tissue.

Biopsy.—The patient was taken to the University of Kansas hospital and laboratory, where I removed a small piece of the gastrocnemius muscle under local anesthesia with a .7 per cent. solution of quinin and urea hydrochlorid. A layer of subcutaneous fat 12 mm. thick covered the muscle fascia. A section about 5 mm. square removed from immediately beneath the muscle sheath contained no muscle bundles, all being substituted by fatty tissue. A second deeper section, about 7 mm. square, contained some pale muscle tissue and much fat. The wound bled freely. Unfortunately the piece placed in a Zenker solution for more careful preparation proved to be entirely fat. The other was sectioned immediately with a freezing microtome. Van Gieson, hematoxylin-eosin and Bismarck-brown stains were employed. Muscle bundles can be seen terminating abruptly in a replacing adipose tissue. Some muscle fibers in process of destruction are seen ending in continuity with substituted connective tissue. There is much fatty tissue between the bundles. No hypertrophied fibers can be found. All are quite uniformly atrophied, measuring 8 to 12 microns in diameter. There is a great nuclear proliferation, the sarcolemma nuclei appearing four or five times as numerous as in the normal. The blood-vessels, including the capillaries, have greatly thickened walls, the adventitia showing the greatest amount of increase. The intima is not involved.

DIAGNOSIS

The diagnosis of amyotonia congenita is made in my case without any hesitancy, all the fundamental symptoms being demonstrated. The hypotonia of a severe grade, the marked general skeletal muscular weakness,

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with soft flaccid muscles, absent deep reflexes, diminution of electrical excitability of muscles, but no reaction of degeneration, no orthopedic deformities, normal mind, normal sensation, no subjective pains, no bad heredity and a general healthy appearance make a strong argument for an undoubted diagnosis. The history regarding a congenital onset is incomplete, but there are a few indications.

In a differential diagnosis several diseases must be eliminated. Poliomyelitis acuta can be excluded by its sudden onset, course, acute febrile period, selection of muscle groups, late trophic disturbances and occasionally musculo-tendinous contractures. Polyneuritis gives pain to pressure and movements, and has a different course and onset. The pseudoparalyses of rickets, scorbutus and Barlow's disease are readily eliminated by their outspoken symptoms, cause and course. In the pseudoparalysis of syphilis we look for other specific symptoms and malnutrition. The Wassermann reaction, or one of its reliable modifications and the diagnostic lumbar puncture should be resorted to where doubt exists. In myasthenia gravis we have a different age, course, electrical findings and bulbar symptoms. Myelitis, hematomyelia and gliosis give afferent tract symptoms. Little's disease with its spasticity is the antithesis of amyotonia congenita. In some cases of imbecility and idiocy there exist much hypotonia and retarded muscular development. The mental defects are evident early, and frequently anatomic stigmata of degeneracy may be found. In cerebellar lesions are found a marked ataxia, normal or slightly increased tendon reflexes and a hypertonia rather than a hypotonia as in amyotonia congenita. The myopathies offer the greatest difficulty in a differential diagnosis. In simple idiopathic muscular atrophy the onset occurs at a different period; there are usually hereditary manifestations, most often similar, a different distribution of the muscular

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weakness, evident atrophies or pseudohypertrophies, no indication of the condition being congenital and not the same hypotonia of amyotonia congenita.

PATHOLOGY

Our knowledge of the pathology is incomplete. The few autopsies and biopsies indicate that the disease is one primarily of the skeletal muscles or of their motor nerve endings. My biopsy shows a great diminution in size of the muscle fibers and marked increase in number of sarcolemma and muscle fiber nuclei. The myoplasm of many of the muscle fibers has undergone a complete degeneration and is replaced with adipose or connective tissue. The blood-vessels show much thickening, especially of the adventitia.

Spiller[6] has reported the first complete autopsy, in which case were found a diminution in size and a hyaloid appearance of the muscle fibers of the calf and back. The transverse striæ were absent. The spinal cord was normal. Smith[11] studied the ductless glands and the hemolymph system in the same case and found the Hassal corpuscles of the thymus gland much increased in size and number. Some of the bodies presented a hyaloid appearance. Oppenheim[12] reflects a doubt about the diagnosis in this case.

Baudouin's[13] excellent post-mortem report shows a slightly arrested development of the anterior horn cells of the spinal cord, spinal roots and peripheral nerves not of an inflammatory nature. He found hypertrophied muscle fibers and many atrophic ones, a sclerosis and a replacement with connective tissue. The blood-vessels were a little thickened. He concluded that it was a regression similar to that found in the myopathies. The thymus gland was sclerosed with a diminished number of Hassal bodies. The thyroid was in a

11. Smith: Univ. Pennsylvania Med. Bull., October, 1905.
12. Oppenheim: Nervenkrankheiten, Ed. 5, Vol. i, Die Poliomyelitis anterior acuta, Differential diagnose, p. 242.
13. Baudouin: Semaine méd., 1907, p. 241.

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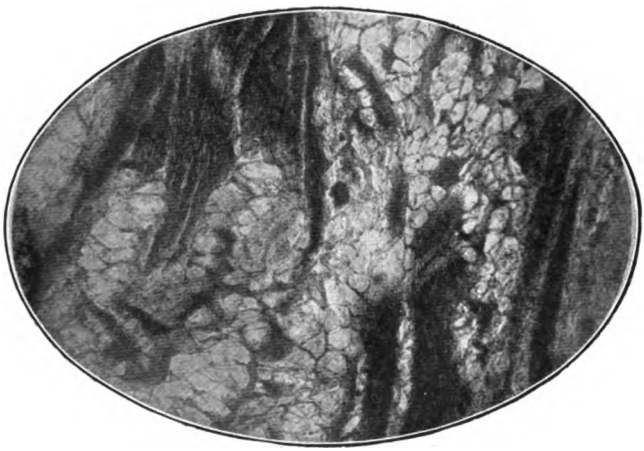

Fig. 1.—Specimen from case of amyotonia congenita. Magnification × 118. Hematoxylin-eosin stain. All remaining muscle fibers atrophic, 8 to 12 microns in diameter. Replacement with fat and connective tissue. Muscle nuclei multiplied several times the normal. The muscle fibers have completely lost their cross-striations.

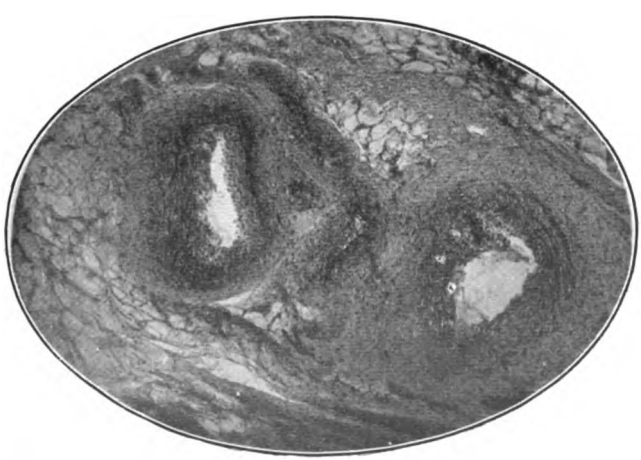

Fig. 2.—Specimen from case of amyotonia congenita. Showing intramuscular blood-vessels with much thickened adventitia and media.

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state of hypofunction with an intense sclerosis, little colloid substance remaining.

Collier and Holmes[9] report one autopsy and one biopsy. They found markedly atrophic and hypertrophic skeletal muscle-fibers, replacement of degenerated ones with fat, muscle nuclei multiplied several times the normal and increased connective tissue. The myelin sheaths of the peripheral nerves were poorly developed. The ventral spinal roots contained fewer fibers than are found in the normal. The anterior horn motor cells were diminished in size and number (1/3). Dorsal horns, Clarke's columns and white columns were normal.

Bing's[14] biopsy report with almost negative findings is incomplete.

ETIOLOGY

The etiology is dark. Heredity certainly plays no rôle as a causative factor. The only exception on record is that of Silvestri,[15] where the heredity was dissimilar. The mother learned to walk late, had a slight goiter and osteomalacia during pregnancy. A maternal aunt had a Landouzy-Déjerine progressive muscular atrophy. One brother had a juvenile progressive muscular atrophy with pseudo-hypertrophies. Two brothers had rickets and one myxedema. Theories of disturbance of internal metabolism or the hemopoietic glands have been advanced, but nothing conclusive has been demonstrated.

TREATMENT

For such an essentially chronic disease as amyotonia congenita too few years have lapsed since our recognition of it to lay out any closely defined plan of treatment, and this holds particularly true in view of the undetermined exact etiology. More than 50 per cent. of these cases can be improved, a few being on record where the improvement has been decided. This is one reason for not allowing them to be classed among the

14. Bing: Med. klin., 1907, i, No. 6.
15. Silvestri: Gaz. d. Osp., 1909, xxx, 577.

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myopathies, most of whose forms continue to steadily decline without interruptions. Hygienic, mechanical and medicinal means of therapy may be resorted to.

All these patients are prone to attacks of acute pulmonary infections. In a large percentage of the reported cases the patients have had bronchitis or bronchopneumonia from which the mortality rate has been high. In 16 per cent. of all the known cases the patients have been registered as dying from pneumonia or bronchopneumonia. My patient had an attack of catarrhal pneumonia from which, after the recovery, there was begun a noted immediate improvement in her motor power, possibly due to the loss of some fatty weight. It is not desirous to encourage the addition of much weight dependent on adipose tissue.

Massage properly applied is valuable. It is perhaps proper to caution against kneading the muscles too vigorously or excessively. If electricity is considered, faradism should be used to those muscles which react to the current, and galvanism to those which do not respond to the interrupted form. Cold baths have been employed, but I would favor the hot should hydrotherapy be indicated. Strychnin in small doses over long periods may be beneficial. Phosphoric and arsenical preparations are worthy of a trial.

CONCLUSIONS[16]

My few concluding remarks shall be confined to a discussion concerning the question of amyotonia congenita being admitted as a distinct disease.

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16. Other literature, not referred to elsewhere, is as follows:
Schüller: Wien klin. Wchnschr., 1904, p. 722.
Hagenbach-Burchardt: Jahrb. f. Kinderh., 1904, p. 47.
Tobler: Jahrb. f. Kinderh., 1907, xiv, 33, No. 1.
Ausset: Bull. Soc. de méd. du nord, 1908.
Gastonquay: Bull. méd de Quebec., 1908-1909, x, 97.
Levi-Sirugue: Gaz. d. hôp., 1909, lxxxii, 173.
Coombs: Brit. Med. Jour., June 15, 1907.
Leclerc: Gaz. d. hôp., 1907, lxxx, 1683.
Variot: Bull. Soc. de pédiat. de Paris, 1907, ix, 246.
Simonini: Riv. di. clin. pédiat., 1907, v, 845.
Smith: Univ. Pennsylvania Med. Bull., October, 1905.
Sorgente, P.: Due casi di atonia musculare congenita di Oppenheim, Pediat., May, 1906.
Iovane: Pediat., March, 1906, xiv.
Cattaneo. Bull. della clin., 1906, viii.

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The most likely field for argument is that of the myopathies. In a discussion of this point before the neurological section of the Royal Society of Medicine of London in the spring of 1909, Collier made a strong plea for its entity in the list of classified nervous diseases. Batten vigorously opposed this position, maintaining that it should be considered as one of the myopathies. I see no other location for this rare neurological condition excepting among the myopathies if we refuse to allow it to stand by itself.

Bing's[14] case, in which a biopsy was performed, showed little or no muscle changes, which prohibits it from being classified as either amyotonia congenita or a myopathy. The description sounds like that of a mongolian imbecile.

Oppenheim[12] in his most recent text-book on nervous diseases has treated the malady in a curious manner, a short space being devoted to the subject under the differential diagnosis of poliomyelitis acuta.

Unfortunately as yet we cannot turn to the pathologic findings to support the contention for placing this group of cases in a class by themselves. The strong support from clinical findings gives us sufficient encouragement to allow the disease to have a real existence.

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THE RELATION OF STUTTERING TO AMUSIA

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I have elsewhere[1] defined stuttering as "a condition in which, through lack of coordination of the nervous mechanism controlling the organs of speech, which may include either excessive or deficient innervation, there is a difficulty in enunciation which may comprise either spasmodic effort without articulate sound, or frequent repetition of the same articulate sound before the utterance of the one following. With it may be associated compensatory spasm of muscles not directly involved in speech." While this definition, an adaptation of those of others, seemed satisfactory to me at the time, I have since come to realize that like those of others, it is simply an enumeration of symptoms. To my mind there has never been a satisfactory explanation of the cause of stuttering. To treat it as a neurosis, "a mental tic," is not going far enough. A neurosis it undoubtedly is, after the establishment of the compulsive idea, but calling it such does not explain why certain individuals under the influence of fright, imitation, acute disease or nasopharyngeal irritation, begin to stutter while others equally neurotic and exposed to the same influence do not.

ETIOLOGY

The observations of Scripture[2] emphasizing the fact that practically all stutterers habitually speak in a mono-

1. Stuttering in School Children; Its Prevalence and Treatment, St. Louis Med. Rev., August, 1909.
2. Scripture, E. W.: Treatment of Stuttering and Stammering, Med. Rec., New York, March 21, 1908.

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tone, are most important, not only from the standpoint of treatment, but also as having a bearing on the etiology. That the monotonous voice is a factor in the causation of stuttering does not at all conflict with Claiborne's suggestion[3] that stuttering should be classed among the conditions caused by congenital defects in the centers controlling speech. In a paper[4] read a short time ago before the National Association for the Study and Education of Exceptional Children, I placed stuttering among the group of developmental defects caused by biologic variations in centers and commissures through which are derived perceptions of music and rhythm. If we accept the theory held by Edgren and others that the different forms of amusia are dependent on damage to special centers and commissures which are adjacent to, but not identical with, those whose damage gives rise to the corresponding forms of speech defect, we must assume that an auditory center for tones will correspond with the auditory word-center, a kinesthetic tone-center with the kinesthetic speech-center and a visual center for notes with the visual word-center.

According to Bastian,[5] "the existence of tone-deafness as an isolated and inherent defect is probably far from rare." There are persons who are incapable of discriminating between the two notes of an octave and who notwithstanding the most frequent opportunities of hearing music remain quite incapable of distinguishing one tune from another. Variations in the kinesthetic center or its commissures would account for the inability of some individuals to "carry a tune." There are some persons who, while possessing a "musical ear," are still unable to reproduce what they have heard. I have, on a previous occasion,[4] drawn attention to the connection between amblymusia and idioglossia.

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3. Claiborne, J. Herbert: THE JOURNAL A. M. A., April 18, 1908.
4. Biological Variations in Higher Cerebral Centers Causing Retardation. Paper read before The National Association for the Study and Education of Exceptional Children, April 21, 1910.
5. Bastian, H. Charlton: Aphasia and Other Speech Defects, p. 295.

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Nature is all curves and harmony. We are constantly receiving, through the special senses, impressions suggestive of modulation, inflection and consonance. On the visual center are registered impressions of form derived from the contour of hills and sky, from the flight of birds, the waving of the branches of trees, the falling of leaves; on the auditory center, from the sound of running water, the soughing of the wind, the rustle of leaves, the songs of birds; on the centers controlling the sense of touch are registered the impressions received from the handling of smooth and rounded objects, and on the general kinesthetic centers the impressions of rhythmical movements made in walking, running, bending, climbing, breathing, etc.

It seems therefore not unreasonable to suppose that these various impressions, registered as they are, in areas of which the centers concerned in speech are a part, have much to do not only with its development, but also with the proper control of the nervous mechanism of the speech-forming organs. To state more clearly, it would appear that impressions of modulation, inflection, consonance and rhythm are registered on special centers which are a part of the general visual, auditory and kinesthetic areas, and that each of these beside being in direct relationship with the word-center of the particular area of which it is a part, is also in commissural relationship with the corresponding centers of other areas.

I further believe that the monotonous voice of the stutterer is the result of a biologic variation in one of the music centers or its commissures, most likely, in the kinesthetic center or the commissure between it and the auditory music-center. It may be held that not all those with unmelodious voices stutter. While they may not do so habitually I believe it will be found that they will sometimes stutter, especially when laboring under strong emotion or fatigue, or when in poor physical condition. The child does not stutter from the time he begins to

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speak but if he is the possessor of a monotonous voice he will do so as soon as he is subjected to one of the exciting causes mentioned above or to reflex irritation from any source, be it eye-strain, phimosis, adenoids, or worms. The compulsive idea then becomes fixed. I have been asked why, if stuttering bears the relation to amusia which I claim it does, many stutterers are able to sing. I think this fact can be explained on the same basis that Dercum explains the same ability in some cases of aphasia. He says:[6] "The curious instance of the singing aphasics can only be met by a similar explanation (reenforcement). For instance, I had under my care some years ago a Broca aphasic, whose speech was limited to "yes," "no" and a few brief expletives. He could, however, sing—among other things—"Auld Lang Syne," carrying the air well and enunciating each word clearly. No other explanation, it would seem to me, than that of reenforcement overcoming inhibition can apply in such a case."

METHODS OF TREATMENT

If we accept the theory that stuttering is due to biologic variations in centers which are a part of or are closely associated with centers making up the zone of language, the reason for the success and failure of many empirical methods of treatment of this disease are apparent. Practically every method that has been suggested is based both on distraction from the compulsive idea and on oft-repeated impressions of correct speaking. In other words, the patient receives from his instructor auditory and visual impressions of properly inflected and articulated words, and further than this by repetition educates his own brain cells, assisting them to perform their proper function in unison with other cells, or by forcing the opposite hemisphere to supply a center similar to that which is imperfectly developed.

6. Dercum, F. X.: The Interpretation of Aphasia, Am. Jour. Med. Sc., November, 1909.

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This is done principally through the glosso-kinesthetic center acting in conjunction with the various music-centers. Failure to cure and relapse after apparent cure are due to development of the glosso-kinesthetic center, with failure to bring about functional relationship with the music-centers. The "melody cure" advocated by Dr. Scripture fulfills all the indications necessary for a complete cure beyond danger of relapse. It should, however, be persisted in until every assurance is felt that the centers controlling melodious speech have assumed the necessary state of activity. Every effort should be made to imprint musical impressions through every available avenue, and to allow every possible opportunity for their expression. It is a well-known fact that music, dancing, rhythmical movements, drawing, etc., are great aids in the development of speech and of general cerebral activity in the backward and feeble-minded.

Another great aid in the treatment of this class of cases is the reversal of dexterity as advocated by Claiborne.[3] It is generally conceded that in right-handed individuals the complicated mechanism of speech is controlled by the zone of language in the left cerebral hemisphere.

"It must needs be admitted that there is a general auditory, a general visual area, and a general kinesthetic area in the right hemisphere, and that incoming stimuli make on it an impression similar to that which they do on the so-called "educated" hemisphere. These impressions are bilateral in reception, but unilateral in interpretation. This unity of interpretation is determined by commissural fibers of the corpus callosum. Now the same factors that determine right-handedness determine also that the left hemisphere shall be the executive speech side, but the elementary work is done on both sides."[7]

7. Collins, Joseph: Ref. Handb. Med. Sc., 1, 419.

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Now as Claiborne says,[3] "It is in the highest degree unlikely that a congenital defect should occur on both sides of the brain and in the same place."

In a biologic variation in brain-centers not so much can be expected from restitution of non-functionating cells as from a process of functional compensation, brought about by the undamaged hemisphere gradually taking on new or greatly developed functions.

General kinesthetic impressions have a decided influence on the development of the speech-centers as is proved by the marked improvement shown by cerebral hemiplegics after tenotomy. The more frequent and dexterous use of the muscles of one side would, therefore, assist materially in the development of the speech-centers of the opposite hemisphere.

I have reported two cases of congenital word-blindness in boys both of whom were originally right-handed who learned to read after they had become proficient in the use of the left hand together with special training. In one,[8] a youth of twenty, who stuttered very badly, I noticed a decided improvement in speech soon after he had begun to use the left hand. In fact, although I had had him under treatment for several months I had been unable to get him to speak with any degree of inflection, until after he had begun the use of the left hand. His subsequent improvement in speech was very rapid. It has been my custom for the past year, in addition to giving all my stuttering patients exercises directed toward correcting their various defects of speech, to advise them to use, when right-handed, the left hand in preference to the right. I am fully satisfied of the value of this procedure, and while I do not believe that the reversal of dexterity will in itself correct stuttering or any other developmental defect due to a biologic variation in special cerebral centers, I do believe

8. Congenital Word-Blindness as a Cause of Backwardness in School Children; Report of a Case Associated with Stuttering, Pennsylvania Med. Jour., January, 1910.

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that it will prove itself a very valuable aid when taken in conjunction with other approved methods of treatment.

CONCLUSIONS

While incapable of proof, it is reasonable to suppose:

1. That the defective use of the muscles of inspiration, expiration, and of the lips, tongue and throat, resulting in stuttering is the "result of imperfect coordination, caused by disconnected and erratic discharges from the cortex."

2. That this incoordination is between the nervous mechanism controlling the acts of vocalization and articulation and the centers having for their function the appreciation and expression of melody and harmony, and is due to a biologic variation in such a center or its commissures.

3. That the cure of stuttering is only accomplished by a process of compensation brought about by the education of cells previously non-functionating, and by forcing the opposite hemisphere to supply a center similar to that which is imperfectly developed. To this end the reversal of dexterity would seem to be a reasonable procedure.

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CHOKED DISC IN ITS RELATION TO CEREBRAL TUMOR AND TREPHINING

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When I was invited to be the guest of the Section on Nervous and Mental Diseases of the American Medical Association, and to read a paper before this famous body, I felt highly honored, and it was with the greatest pleasure and thanks that I accepted this invitation. Having leave to choose the subject I did not hesitate long and selected the important theme of choked disc in its relation to cerebral tumor and trephining.

There is hardly any point, which has for so long a time been subject to as much controversy as the pathogenesis of choked disc. In spite of numerous works, experiments, clinical observations and pathological—anatomical investigations, we have not succeeded in bringing about an agreement between the various investigators on this topic. Just lately the old quarrel on the pathogenesis of choked disc has become active again in Germany.

Surgical experience being, to my mind, of great importance for clearing up the origin of choked disc, I was convinced that America would be the best forum for discussing this question, because America possesses a greater experience on the effect of and indications for trephining in cerebral tumor than we do in Germany, thanks to the studies and works of Taylor, Mills, Spiller and Frazier, De Schweinitz, Weisenburg, Putnam, Knapp, James Bordley and also to the new methods

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of Harvey Cushing, not forgetting, however, England's contributions, by Horsley, Gowers, Gunn and Leslie Paton.

The fact that I was an ophthalmologist at first and devoted myself to neurology later has brought it about that I have been occupied for twenty years with studying the changes of the fundus of the eye in diseases of the brain.

By request of Professor Uhthoff of Breslau, I made a report on the pathogenesis of choked disc before the seventy-sixth Versammlung Deutscher Naturforscher und Aerzte in Breslau in 1904. Since that date so much new work has been published, that I deem it necessary to give you a brief outline of the stand we take on this important question in Germany.

REVIEW OF WORK AND THEORIES ON CHOKED DISC

Tuerck was the first, who in 1853, when describing a case of cerebral tumor with retinal hemorrhage expressed the idea, that the changes in the fundus of the eye were due to increase of intracranial pressure. However, only in 1860 the scientific world became acquainted with the so-called choked disc through Albrecht von Graefe's work on complications of optic nerve-inflammation with brain diseases. Von Graefe saw the cause of choked disc in brain pressure, due to cerebral tumor, the sinus cavernosus being thus compressed and the voiding of the vena ophthalmica respectively the vena centralis retinæ being inhibited; bringing about dilatation and twisting of the retinal veins as well as edematous swelling of the optic disc. He explained the inflammatory changes, first, as originating from retinal extravasations of blood; secondly, from the irritating effect of swelling of the optic disc, still increased by toughness of the scleral ring.

Von Graefe's hypothesis, however, soon encountered opposition. Hughlings-Jackson took the optical affection for a reflex symptom, but it was the anatomic

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facts found by Sesemann which principally disproved the theory. It was he who proved that the central retinal vein has an off-flow into the facial veins, quite independent of intracranial pressure. Moreover, Sesemann argued that compression of the sinus cavernosus could not be affected as easily as one might think. Later these statements made by Sesemann in 1869, though accepted everywhere, even by von Graefe himself, have met opposition from the well-known anatomist, Merkel, and from Gurwith, who, on the basis of extensive anatomical investigations, argued that the greatest part of the orbital venous blood flows into the sinus cavernosus. Quite recently J. Tudeich and Krüdener have endeavored to uphold the old theory of von Graefe for those cases of choked disc, in which intracranial pressure is localized.

The second principal theory of choked disc is the one brought forward by Schmidt-Manz's theory of mechanical transportation, based on Schwalbe's discovery of the communication between the intravaginal space of the optic nerve and the intermeningeal space of the brain. According to the views of both these authors a vaginal hydrops of the optic nerve is caused by increase of intracranial pressure, for instance, owing to cerebral tumor, and consequently, incarceration of the intraocular end of the optic nerve would occur, choked disc henceforth resulting.

Von Schulten, by his experiments, confirmed Schmidt-Manz's theory, which was readily accepted, but soon also vigorously combatted.

In 1881 the famous ophthalmologist, Leber, brought forward a new theory of choked disc before the London Congress. According to Leber it is a real inflammatory process, a papillitis, not preceded by any stage of pure edema or pure venous stagnation. The originators of inflammation are in the fluid which has come from the intermeningeal space of the brain into the intervaginal space of the optic nerve. Even though a real hydrops

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of the optic nerve were absent, yet choked disc could occur, because the point is not the quantity of the fluid, but its quality.

Deutschmann supported the theory of Leber by several works and established the hypothesis that the cerebral tumors produce toxins, which call forth inflammation of the optic nerve and its sheaths.

Besides Hughlings-Jackson, Gowers also was of the opinion that intracranial tumors could cause neuritis optica through irritating influences acting as foreign bodies.

In modern times Zellweger and Elschnig, the latter of whom disposed of twenty-eight cases of choked disc, which he examined microscopically, declared themselves for Leber-Deutschmann's inflammatory theory. Elschnig says, that in intracranial tumors the primary is a neuritis of the optic nerve, whereas edema is secondary and only an accompanying symptom.

Liebrecht, in 1902, is likewise of the opinion that the primary manifestations are inflammatory changes within the optic sheaths, in connection with which a stagnation of the lymph occurs in the optic nerve and disc.

We may quote, among other theories, the one of Parinaud. According to him the various intracranial diseases call forth a stagnation in the optic disc only in the case of complication with hydrocephalus internus. With this is connected edema of the brain, which by itself affects an equivalent edema of the optic nerve This may be followed by inflammation—*neurite oedémateuse.* In the presence of edema of the optic disc, according to Parinaud, the diagnosis of hydrocephalus and cerebral edema only is admissible.

Lourdill accepted Parinaud's theory. However, he considered the primary change to be an edema of the ependymalglia, which continues in a direct way in the glia of the chiasm and of the optic nerve.

Also, according to the opinion of Ulrich (1887) cerebral edema propagates into the optic nerve.

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In 1904 extensive researches (forty-six eye examinations) made by Kampherstein from Professor Uhthoff's clinic at Breslau, proved that the most invariable symptom consisted in an interfasciary edema of the optic nerve. For this reason in most cases choked disc is nothing but an edema propagated from the brain, penetrating into the optic nerve, and through it into the optic disc by the way of the lamina, creating hereby incarceration of the intraoculary optic nerve-end a swelling within the rigid scleral ring.

Benedict and Adamkiewicz occupy a position apart with regard to the pathogenesis of choked disc. The former believed the vasomotor nerves to be irritated by the tumor, thus causing an inflammation of the optic disc by reflex. The latter regards the choked disc as the result of a neuroparalytic inflammation due to irritation of the trophic centers proper to the optic nerve.

Knapp, Deyl, von Gross and Yamaguchi published cases to prove the causation of choked disc through compression of the central vein.

PRESENT-DAY THEORIES

In our days we seem to be reaching a compromise. Thus Greeff expressed himself as follows: "It seems to be the case that stagnation of the cerebrospinal fluid plays an important rôle, but that inflammatory irritations are concurring."

In 1907 I published a treatise, in which I pointed out again energetically my views, which I had presented in 1904, viz., that a palliative operation should be performed in cases of inaccessible brain tumor in order to avoid threatening blindness.

SUMMARY OF CASES

I disposed of nineteen cases. In two of these choked disc decreased only after the trephining hole had been enlarged, allowing a great outflow of cerebrospinal fluid. In two cases decompression had no effect what-

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soever as far as the choked disc was concerned. These were cases of extraordinarily large, rapidly increasing brain tumors. In one case only, a tumor of the base. the operation was followed by very high fever and rapid exitus. No doubt it must have been one of those cases which have just been described by Dr. George J. Jacoby in New York, because post-mortem examination did not explain the cause of the hyperthermia.

In all the rest of the cases the effect of decompression on choked disc was obvious.

In one case I was able to observe, as did Cushing in two cases, a decrease of choked disc directly after decompression.

I apply this term in the sense of Frazier, as he explained in an address before the Toronto Academy of Medicine last year. He said: "The term decompression implies not simple trephining, but the removal of the dura.

DECOMPRESSION OPERATION

In all my cases decompression has been done in this sense. Often I could observe that the choked disc decreased in the measure in which the outflow of cerebrospinal liquor increases. As a rule it takes several weeks or months to flatten down.

In order to obtain good results as far as vision is concerned it is imperative to operate early, viz., while the visual power is still of use. In some cases I have seen vision improve considerably after decompression.

E. von Hippel, in his studies of the literature in 1908, arrived at the same result. He does not, however, give his personal experience.

Philip Coombs Knapp, in 1906, published a vast number of cases in which no tumor was found by operation or in which decompression alone was done. Knapp affirms that the development of choked disc is arrested only in about half the cases. I regret very much that Dr. Knapp is not present. I would ask him whether he

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took any notes of the degree of visual power and time of operation. This is the important point.

I indicated as being the best time for operating the very beginning of visual diminution. If the operation is done after this epoch, atrophy of the optic nerve very easily develops, as I have seen several times.

The place to trephine is obviously that point of the skull, under which the tumor is supposed to be.

In case a local diagnosis is impossible I advised trephining on the right parietal lobe. Recently I had the pleasure of conferring with Harvey Cushing in Baltimore about this question, and he had the kindness to show me several cases of decompression by his subtemporal method, and I must say that seldom have I seen such good results, so that I here gladly say that in the future I shall advocate this region for decompression and not the right parietal lobe, as I have formerly recommended, although realizing that the subtemporal operation, though preferable, may be more difficult to perform.

INOPERABLE TUMOR

What shall we do in such cases, in which an osteoplastic resection after the method of Wagner has been made and an inoperable tumor or a very high tension of brain without surface change is found? The new method of Cushing is very ingenious in combining exploration with subtemporary decompression, as he has described in the transactions of the American Surgical Association.

In subtentorial cases trephining is not so dangerous in my experience as it was thought before.

Spinal puncture, puncture of the lateral ventricles and the so-called Balkenstich procedure (Braman) cannot possibly, in my experience, compare with trephining of the skull as regards results. My conclusions were summed up as follows: Palliative trepanation of the skull according to the modern standing of surgery, in

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the hands of a skilful surgeon who has experience in neurological work is almost without danger and an extremely beneficial operation, which I should recommend in every case of inaccessible brain tumor, in order to ease the torments of the patient, and above all, to save him from impending blindness.

This standpoint expressed by me first in 1894 and defended in 1902 against the opinion of Bergmann before the Surgical Congress at Berlin, has met with such universal favor in America, that it seems superfluous to mention it specially, as Horsley, Cushing, and others expressed themselves in the same way.

INCREASED TENSION THE CAUSE

These extensive surgical experiences, however, possess a great value for the conception of the pathogenesis of choked disc. They certainly admit of the conclusion that the main point in bringing about choked disc consists in increased cerebral tension. For in the majority of cases decrease of tension by trephining is followed by disappearance of choked disc, and so quickly, too, at times, that toxins could not possibly have had any influence; particularly in those cases in which the dura has not even been opened.

The subtle process in the genesis of choked disc has remained occult up to date in spite of plenty of experiments and microscopic-anatomic research.

RESULT OF AUTHOR'S INVESTIGATIONS

I have myself, during the latter years, devoted much time to pathologic-anatomic examinations of choked disc in brain tumor, which we meet with frequently in the Hospital St. Georg at Hamburg.

During these investigations I could also state the fact that in some cases of choked disc a dilatation of the intervaginal space has been missed. Moreover, I have often seen cases of ampulla nervi optici without choked disc. In many cases I found only slight microscopic

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changes in the optic nerve and in the sheaths, though choked disc was exquisitely pronounced. In other cases, however, I found the space between the fibers of the optic nerve had become wider, allowing the conclusion of the presence of edema, but I was not able to prove it. To my mind the methods heretofore in use for examining choked disc are entirely insufficient, all the more as it is essential to show the presence of edema.

Considering what manipulations must be performed with the optic nerve, more or less bruising it in its removal, it is quite obvious that the results of microscopic examinations, often modified by artificial products, must often give quite varying pictures, which certainly cannot render the subtle conditions *in vivo*. Already the preparation for embedding the nerve in celloidin, and letting it rest in alcohol of increasing percentage proves altogether, that the optic nerve is being exposed to considerable shrinking processes, altering entirely the conditions which the supposed edema is expected to have caused.

I am trying at present new methods, which should avoid this faulty treatment. At present I am, however, not in a position to report on the results obtained.

I should like to add concerning my microscopic examinations, that in some cases I found ectatic capillaries and even hemorrhages in the optic nerve as well as in the dura. So far as inflammatory changes are concerned, I found in some cases cellular infiltrations between the sheaths, which would have been interpreted according to former views as inflammation. According to the opinion of competent pathologic-anatomic authorities (Prof. Fraenkel and Prof. Simmonds, of Hamburg) these were proliferations of endothelial cells, and therefore not real inflammation. In this way probably the diverging findings of the authors must be understood, some calling inflammation, what others describe as an increase of endothelial elements.

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CLINICAL OBSERVATIONS

Returning to the clinical observations, at present I dispose of twenty-six cases in which decompression has been performed for cerebral tumor.

Seven were cases of tumor of the cerebellum. In one of them a cyst was found, after removal of which the patient got well. Three were cases of tumor of the acoustic nerve (Kleinhirnbrückenwinkeltumoren.)

One was a tumor of the parietal lobe. In this case there occurred a hernia, as was described later by Cushing, who recommended the establishment of hernia as a decompressive measure for inaccessible brain tumors. The patient in this case continued to live for nine months with good visual power and in good general condition.

Three were tumors of the frontal lobe. In one of the patients there was choked disc in one side only, namely, opposite to the seat of the tumor. Three were cases of tumor of the base. In these cases decompression was of no avail.

In two cases of cerebral tumor decompression only had the advantage of removing headache and vomiting. The patients had become blind already before the operation.

In a very remarkable case in which the patient was operated on for supposed tumor of the cerebellum (choked disc, cerebellar ataxia, abscence of patellar reflexes, occipital headache, vomiting) the result of decompression was very deplorable. The patient died of hemorrhage immediately after and post-mortem examination revealed the total absence of any tumor and the presence of chronic meningitis. A similar observation has been quite recently published by Oppenheim, in Berlin.

No less remarkable are the five following cases: All of the patients presented the symptoms of a brain tumor: choked disc, headache, vomiting; two of them had focal symptoms of hemiparesis of the right side. One showed the symptoms of a tumor of the acoustic

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nerve. When decompression was performed, there was no tumor found. After trephining and outflow of cerebrospinal fluid the choked disc disappeared and brain tension and its symptoms were effaced.

The first patient, 27 years old, was operated in 1899 and was presented by me before the Surgical Congress at Berlin in 1902. He now is doing very well, with a slight diminution, though, of visual acuteness. He is busy in a banking house from morning to night.

The second patient, operated in 1904, was a teacher in drawing, who was still active until 1909, when actually pensioned off.

The third patient is a boy twelve years old, who had totally lost his sight under the symptoms of brain pressure and choked disc. He was trephined over the parietal lobe with removal of the dura after the method of Wagner in 1909. The boy is absolutely well and one of the best scholars in his class.

The fourth case concerns a man aged 22, with the symptoms of a tumor of the central lobe. Nothing was found during the operation; all symptoms receded, leaving, however, a slight weakness of the right hand. He is now fulfilling the duties of a watchman.

The last case, a man of fifty-six, is also active in performing his business. There was no decompression made, but a puncture of the left lateral ventricle relieved him of all symptoms of brain pressure. Only a slight weakness of the right hand remained.

THE QUESTION TO BE ANSWERED

The question arises in these cases: Have we to deal with a tumor, which has become regressive, or a so-called pseudotumor, such as was described first by my colleague, Dr. Nonne, of Hamburg, or else hydrocephalus internus, or finally Reichardt's cerebral swelling?

In the present state of science it is impossible to answer these questions definitely. Let us hope the time

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is not far off when by the united work of the neurologists of the New and the Old World this high aim will be achieved!

ABSTRACT OF DISCUSSION.

MR. J. HERBERT PARSONS, London, Eng.: I hold that the pathogenesis of the disease is a question of edema and not of inflammation. I suppose that we are all absolutely agreed as to the importance and utility of the relief of intracranial pressure in the treatment of these cases, whether the tumor is a so-called operable tumor or not. Of course the important point is the relief of the visual symptoms; and here I speak entirely from the pathologic point of view, and therefore perhaps in a manner not altogether acceptable to the neurologists. I do think the importance of determining the time at which interference should take place and the advisability of its interference in these cases, should be determined to a very considerable extent by the opthalmologists. The fundus conditions are ones with which the opthalmologist is very much more familiar than the neurologist, and the opthalmologist can give the neurologist information which is of the utmost importance in assisting him to determine the best time for interference to take place.

I know from many cases that I have seen in England that interference is frequently delayed much too long from the point of view of vision, and in a very large proportion of these cases (a far larger proportion than is at all desirable) the only palliation that can be secured is to give these patients vision during the remainder of their lives, and also to relieve their headaches, etc. That is an enormous boon to the patient. It does not do very much good to interfere in the very last stages.

DR. JULIUS GRINKER, Chicago: The question of whether choked disk is caused by an inflammation or an edema has recently occupied the neurologists of this country, and discussions of this problem have been reported in the journals. Cushing's experiments on animals and his observations on patients before, during and after operation seem for most of us to have settled that point. Others, however, have disputed the contention that edema or mechanical causes are responsible for the so-called choked disk.

About six weeks ago I was consulted in a case in which I made a diagnosis of frontal abscess. In addition to other symptoms, there was a hazy condition of the fundi oculorum which was verified by an opthalmologist. An operation was performed, the abscess was drained and apparently completely evacuated. Two or three weeks after the patient had been considered well, however, I was asked to see him again, and to my great surprise, I found a typical choked disk—a finding

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also verified by an opthalmologist. Three weeks later the same patient developed more symptoms of brain-pressure. He was again operated on but this time the surgeons could not find the seat of the abscess, although it was suspected that another abscess had formed which was the cause of the choked disk. The abscess had just been missed, the patient died, and an autopsy disclosed a cistern of pus connected with the former abscess by a canal no larger than the size of a goose-quill,—the whole constituting a so-called hour-glass abscess.

This case certainly leads to the conclusion that the accumulation of pus back of the first abscess was the actual cause of the choking of the disk, which had been only incipient when the small abscess was present, and it gives support to the mechanical theory of the causation of choked disk. I believe that it is only by clinical experiences of this kind, by experiments as performed by Harvey Cushing and others, and by observations of cases before and after decompression, that we can come to anything like a satisfactory solution of the question of the nature of so-called choked disk.

Dr. J. Bordley, Jr., Baltimore: For several years I have been associated with Dr. Cushing in working at the practical utility of cerebral decompression. So far as the operative result is concerned, it matters little what the pathogenesis of choked disk is; whether it is a simple mechanical pressure which directly forces down the fluid into the optic nerve, or whether it is pressure which results in the production of a toxin. The important fact to determine is whether a decompressive operation relieves choked disk. We know from an experience covering about 300 brain tumors that it does. When the operation was performed without delay, the results were uniformly satisfactory and convincing. The time to operate in these cases is as soon as they are discovered. There is no change in the optic disk with which I am familiar, from the active stage of hyperemia with edema to the later stages of atrophy, in which a decompressive operation is not advisable.

Professor Saenger records a case of a patient who prior to operation was blind but subsequently had complete restoration of vision. It has been my pleasure to see several such cases. We know, of course, that in these cases there was not complete atrophy, and we believe the blindness due to central pressure and not to pressure on the nerve alone.

It is impossible to look at a disk that is choked and determine its exact condition and what may be expected to result from operation. A marked choking with complete atrophy may be unperceived by the observer; or there may be new tissue formation on the disk and in the nerve which so closely simulates complete atrophy that almost any ophthalmologist would be willing to stake his reputation on the diagnosis; nevertheless, when the disk declines and the patient recovers there is perfect vision. I am confident that it

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is not possible to locate the tumor by a study of the disks. While there are suggestive signs they are by no means of positive value. As an example we had a long list, possibly twenty-five or thirty patients, with the greatest choking on the side of the tumor. We were almost persuaded. Then came a series of several cases with the greatest choking on the opposite side. As a rule, however, the greatest choking—or rather the greatest change in the optic nerve—is on the side of the tumor, probably 65 or 75 per cent.

Tumors of the cerebellum, as a rule, give rise to a greater swelling than tumors farther forward. Anatomic variations in the optic nerves make a difference in the degree of swelling on the two sides. Consequently it must be expected that tumors, even of the same histologic structure, will result in a difference in degrees of swelling in different individuals. In our experimental work on animals, where the choked disk was produced with simple saline solutions, the greatest choking usually occurred on the contralateral side. My impression is that the same rule will hold good in patients with intracranial injuries in whom there is hemorrhage. Wherever there was solid pressure, such as produced by paraffin, the greatest change was usually on the same side.

One thing is absolutely certain: a decompressive operation should always be performed when one is dealing with a stationary or increasing choking of the optic disk. There is no excuse for sitting by and waiting for vision to decline; every bit lost may be gone forever, for one may have waited for the beginning of a very rapid end. And it is well to remember that if the sight is not obliterated by pressure, it may be by the new connective tissue which forms in the nerve and in the disk—as the result of long irritation. Like other scar tissue this will shrink, and as it shrinks it snuffs out the life of the nerve.

Dr. A. Saenger, Hamburg, Germany: I am much gratified that my paper has elicited such a lively discussion, and, above all, that Dr. Parsons, the eminent ophthalmologist of London, agrees with me. He declares that choked disk is not an inflammation but an edema of the optic papilla. This statement is very important as coming from a very competent authority on pathologic anatomy in the field of ophthalmology, and also as coming from the English school. We all know how tenaciously the conception of the inflammatory nature of choked disk has been maintained in England. Even Gowers, that well-known authority, has championed this view.

I hope that now there will be a general acceptance of the view that the increased pressure on the brain is the main factor in the origin of choked disk, manifesting itself in the edema of the papilla. There may be inflammatory changes besides, but they are of secondary nature. The inflammatory theory of choked disk, as Leber and Deutschmann propounded it, can no longer be maintained. I am familiar with Dr. Bordley's work

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on the subject, done in collaboration with Cushing. I visited the famous Johns Hopkins Hospital at Baltimore a few days ago and was amazed at the extraordinary amount of material in the field of brain surgery which it at Harvey Cushing's disposal there. I had also an opportunity to see this master in his special field of science operate. I admired the originality of his technic, the position he gives the patient and his extreme caution and conscientiousness in the execution of the operation. We stand on the same ground in respect to early trephining with a tumor in the brain. Cushing, however, goes beyond me in this. He trephines even before actual choked disk has developed, while I prefer to wait until with existing choked disk the vision begins to show impairment.

My warning to refrain from operating too early is based on my last series of five cases, on the latest experiences with pseudotumors, swelling of the brain, chronic meningitis and brain syphilis with negative Wassermann reaction. The deceptive cases of brain syphilis, which so often runs a disguised course, should be constantly borne in mind, and specific "mixed" treatment should invariably be given a trial before any operation on the brain. I have occasionally encountered unilateral choked disk, far more frequently on the side corresponding to the tumor, but still, though rarely, sometimes on the opposite side. I am persuaded that individual changes in the optic foramen are responsible for the lack of development of choked disk. In one case I was able to determine this to be the fact, although it is technically a difficult matter to demonstrate beyond cavil this narrowing of the optic foramen. It is to be hoped that some research worker may turn his attention especially to this question ere long.

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PATHOLOGY AND BACTERIOLOGY OF ACUTE ANTERIOR POLIOMYELITIS *

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AND

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MINNEAPOLIS

I. *PATHOLOGY*

By H. E. Robertson, M.D.

Our knowledge of the pathology of anterior poliomyelitis dates from 1863, at which time Cornil[1] published the report of an autopsy on a patient who died at the age of 49, forty-seven years after the acute attack. He noted the loss of nerve-cells and atrophy of the affected muscles. Two years later Prevost[2] described an autopsy in a still older case in which he found loss of nerve-cells, overgrowth of neuroglia and asymmetry of the cord, with reduction in the size of the left ventral horn.

Johnson and Clarke[3] in 1868 from a study of the spinal cord of a man aged 32, who had suffered his acute attack in early infancy, advanced the opinion that atrophy of the nerve-cells is the cause of changes in the voluntary muscles. A similar case led Charcot and Joffroy[4] in 1870 to state that the ventral nerve-cells are

* Joint studies in the laboratories of the Minnesota State Board of Health and the Department of Pathology and Bacteriology of the University of Minnesota, under the direction of Dr. F. F. Wesbrook.

1. Cornil: Compt. rend. Soc. de biol., 1863, p. 187.
2. Prevost: Compt. rend. Soc. de biol., 1865, p. 215.
3. Johnson and Clarke: Med.-Chir. Tr., London, 1869, ii, 250.
4. Charcot and Joffroy: Arch. de physiol. norm. et path., 1870, iii, 134.

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first and specifically involved, as in progressive muscular atrophy, but more suddenly. These conceptions were based entirely on examination of old healed lesions, with no reported observations of initial stages, and yet for many years they were widely quoted in favor of the theory that the disease is essentially a primary degeneration of the anterior horn cells.

The following year, 1871, Roger and Damaschino[5], after a careful review of the literature to date, reported three cases in children with autopsies, two, six and thirteen months after the onset of the disease. From these studies they concluded that the primary lesion in the cord was vascular and the process an inflammatory softening, of the nature of a myelitis.

The first report of an autopsy on an adult patient, a woman of 67, was published by Gombault[6] in 1873. Death occurred seven years after the acute attack. There was involvement of the anterior horns and some degeneration of the anterior nerve-roots. The lesion was not regarded as primarily inflammatory, thus agreeing with the opinion of Charcot. In a case in which the patient died at the age of 23, twenty years after the acute attack, F. Schultze[7] found changes in the lateral tracts and Clarke's columns in addition to the lesions. With Roger and Damaschino he regarded the process as inflammatory in type. Déjerine[8] in 1878 by studies on two cases in children with autopsies supported Charcot's view of the parenchymatous nature of the disease.

In 1883, Archambault and Damaschino[9] in their autopsy found "red softening" in the anterior horns. Two years later Drummond[10] published a description of the cord from a child with death about seven hours after onset. The ventral horns were congested and showed

5. Roger and Damaschino: Gaz. méd. de Paris, 1871, xxvi; also Compt. rend. Soc. de biol., 1871, p. 49.
6. Gombault: Arch. de physiol., 1873, v, 80.
7. Schultze, F.: Virchow's Arch. f. path. Anat., 1876, lxviii, 128.
8. Déjerine: Progrés méd., 1878, v, 423.
9. Archambault and Damaschino: Rev. mensuelle d. mal. de l'enf., 1883, i, 63.
10. Drummond, D.: Brain, 1885, viii, 14.

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minute hemorrhages. Microscopically there was leukocytic infiltration and the nerve cells were swollen, granular, and ill-defined. The following year Money[11] studied two cases with autopsies, and first called attention to the presence of thrombosis in a vessel in the anterior horn.

Rissler's[12] opportunity was exceptional. His material was from five cases, three early (one adult with death on the eighth day), and two late. In his paper, appearing in 1888, he concludes that in acute cases there is cellular infiltration and degeneration of nerve-cells, but, with Charcot, he believes the nerve-cell changes are primary.

Marie[13] advanced the opinion that an infectious embolism or thrombosis in one of the branches of the anterior median artery may be the starting point of the disease.

In 1893 Goldscheider[14] published the best review of the literature to date. Contrary to Charcot he believes that the primary condition is a congestion of the central arteries of the cord followed by diapedesis and infiltration of the surrounding nervous tissue by small cells and serum. This choking of the gray matter by inflammatory products causes impoverished nutrition and actual disintegration of the nerve-cells of the anterior horn, with ensuing permanent destruction of the nerve-tissue. He suggests that in acute anterior poliomyelitis substances giving rise to cell proliferation affect the walls of the blood-vessels by filtration and diffusion from the blood. In this connection Woodhead[15] points out that the endothelial cells of the cerebrospinal system of capillaries are extremely delicate and active; the lymph flow is great; and if the products of bacteria, or bacteria themselves, have any effect on the walls of the blood-vessels they should manifest themselves here, and

11. Money, Tr. Path. Soc., London, 1884, xxxv, 45.
12. Rissler, J.: Nord. med. Ark., 1888, xx, 1.
13. Marie: Leçons sur les maladies de la moelle, Paris, 1892.
14. Goldscheider: Ztschr., f. klin. Med., Berlin, 1893, xxiii, 494.
15. Woodhead: Williamson's Diseases of the Spinal Cord.

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we should expect to find well-marked vascular and perivascular interstitial changes. Owing to the greater blood supply the toxin will be brought into closer relationship with the gray matter than with the white.

The following year Siemerling,[16] from studies of two cases in infants, with autopsies, stated that, "in the pathogenesis of infantile paralysis, the inflammatory lesion of the interstitial tissue with a distention of the anterior spinal arteries plays the chief rôle. A primary inflammation of the ganglion cells in the sense given by Charcot is not to be admitted."

Bruins and Windschied[17] conclude that the disease is an interstitial myelitis of the gray matter of the anterior horns, having its origin in the blood-vessels. Starr[18] apparently also supports this view.

In 1898, Greene, Wilson and Rothrock[19] reported an autopsy on a woman aged 20, who, after a five days acute illness, with rapid ascending paralysis, showed involvement of the respiratory muscles, and for forty-one days was kept alive by artificial respiration. They believed that the pathologic process was an ascending inflammation of the gray matter of the anterior horns, and extravascular in origin.

Taylor,[20] in 1902, published an important paper on "Poliomyelitis in the Adult," giving the report of one case coming to autopsy, and a careful review of the entire literature. He concludes that poliomyelitis is an infection of undetermined character limited to the region of the ventral horns of the cord, and that the pathologic alterations found are consistent with the theory of primary inflammation acting in the general distribution of the arteries of the cord and a secondary destruction of nerve-cells.

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16. Siemerling: Arch. f. Psychiat., 1894, xxvi, 267.
17. Bruins and Windschied: Twentieth Century Practice of Medicine, xi, 688-9.
18. Starr: Allbutt's System of Medicine, viii, 196.
19. Greene, Wilson and Rothrock: Philadelphia Med. Jour., December, 1898.
20. Taylor: Jour. Mental and Nervous Diseases, 1902, p. 449.

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Two years later Batten[21] reported careful studies of the material from an autopsy on an acute case. From these he forms the opinion that the pathologic changes in the anterior horns are due to a "primary thrombosis of a branch or branches of the anterior spinal artery supplying the gray matter. Such thrombosis may be produced by many and various forms of infection, or, in other words, the disease is not due to a special specific infection."

Bruining[22] is one of the few recent writers who supports Charcot's views. His conclusions are based on material from a case coming to autopsy two years after the acute attack. He found the vessels congested, but no perivascular infiltration. From the absence of any inflammatory changes he believes the disease attacks the nervous elements primarily.

Hock[23] studied the tissues from an acute case, the patient dying thirteen weeks after the onset. He summarizes his conclusions as follows:

1. **Anterior poliomyelitis is the result of a primary inflammatory disease of the blood-vessels of the cord, which may be thrombotic or embolic.**
2. **The destruction of the ganglion cells is secondary and depends in part on the deficient blood supply of the diseased area and in part on pressure and toxins.**
3. **The inadequate collateral circulation within the anterior horns is favorable for sluggish circulation.**

Wickman[24] in 1905 published an extensive monograph. His pathologic material was from nine cases, seven recent and two late. He believes the process is an infiltrative disseminated myelitis, the infiltration lying in close connection with the blood-vessels. He found no evidence of embolism or thrombosis. While the interstitial and parenchymatous changes ran about a parallel course, ganglion-cell degeneration alone was not ob-

21. Batten: Lancet, London, Dec. 20, 1902, p. 1677.
22. Bruining: Ztschr. f. Nervenheilk., 1904, xxvii, 269.
23. Hock: Jour. Mental and Nervous Diseases, September, 1905, p. 545.
24. Wickman: Studien über Poliomyelitis acuta, Berlin, S. Karger, 1905.

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served, but often he found normal cells in the neighborhood of altered vessels. He is inclined to regard the mode of infection as lymphogenous and suggests that it seems probable that the pathologic condition found in poliomyelitis may be due to a poison similar to that of rabies.

Writing in Osler's "Modern Medicine," Buzzard[25] summarizes the changes as follows:

1. Congestion of all blood-vessels of the cord and packing of the adventitial sheaths and lymph-channels with cells.
2. An intense proliferation or exudation of cells limited almost entirely to the gray matter of the anterior horns.
3. Round-cell infiltration of the soft meninges.
4. Retrograde (secondary) changes in those nerve-cells and fibers which are intimately involved in the areas of inflammation.

By far the most important contribution to this subject has been made by Harbitz and Scheel[26] in a monograph published in 1907, in which they report the results of studies on material from nineteen autopsies, grouped as follows:

(a) Thirteen cases of acute poliomyelitis with death from two to ten days after onset.

(b) Four cases with death from one and a half to twenty-two and a half months later.

(c) Two cases of acute encephalitis.

They believe that the disease begins as a primary inflammation of the pia mater extending into the cord and producing a diffuse infiltrating inflammatory lesion closely related to the blood-vessels and chiefly in the gray matter of the anterior horns. They describe the cellular infiltration as consisting "partly of small and large mononuclear lymphocyte-like cells, especially in the pia mater and white substance, and partly of polymorphonuclear leukocytes, particularly in the gray substance." The degeneration of the ganglion cells was

25. Buzzard: Osler's Modern Medicine, Philadelphia and New York, vii, 261-62.

26. Harbitz and Scheel: Pathologische-anatomische Untersuchungen über akute Poliomyelitis und verwandte Krankheiten von den Epidemien in Norwegen 1903-1906, Christiana, 1907.

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marked, their remnants often being surrounded by heaps of leukocytes (neuronophages). The inflammation often had a hemorrhagic character, especially in the anterior gray horns. No mention is made of thrombosis or embolism.

B. Sachs[27] at the last meeting of the Association of American Physicians emphasized the toxic character of the disease-process, its vascular origin and its tendency to involve the whole cord, and yet to expend its chief force on the ganglion cells of the anterior horns. He notes the evidence of sepsis in liver, spleen and thymus and the tendency to bronchopneumonia.

In this review of the literature on the pathology of poliomyelitis we have found records of about 100 autopsies, nearly one-half of which have been performed on old cases.

REPORT OF CASES

Our own studies include material from six autopsies performed on cases occurring in the Minnesota epidemic of 1909.[28] They will be considered in the order of their respective ages.

CASE 1.—Baby boy, aged 9 months. Acute febrile attack followed by flaccid paralysis of upper and lower extremities. Death on fourth day.

Autopsy.—Aug. 10, 1909, nine and one-half hours postmortem. Body is that of a normally developed, well nourished male infant. Peritoneal, pleural and pericardial cavities normal; heart muscle pale and soft. There is a minute slit-like opening in foramen ovale. Lungs in the dependent portions are dark in color and heavy with congestion. Anterior inner margins expanded and feathery. No evidence of pneumonia. Spleen is soft, swollen and dark in color. On section, pulp scrapes away readily, lymph-nodules are prominent and surface deeply congested. Liver shows cloudy swelling. Pancreas and stomach normal. Mucosa of intestinal tract shows several widely separated areas of deep congestion. Adrenals

27. Sachs, B.: Pathology of Poliomyelitis, THE JOURNAL A. M. A., May 28, 1910, p. 1807.

28. These studies have been made possible through the courtesy and cooperation of attending physicians and associated medical and health officials.

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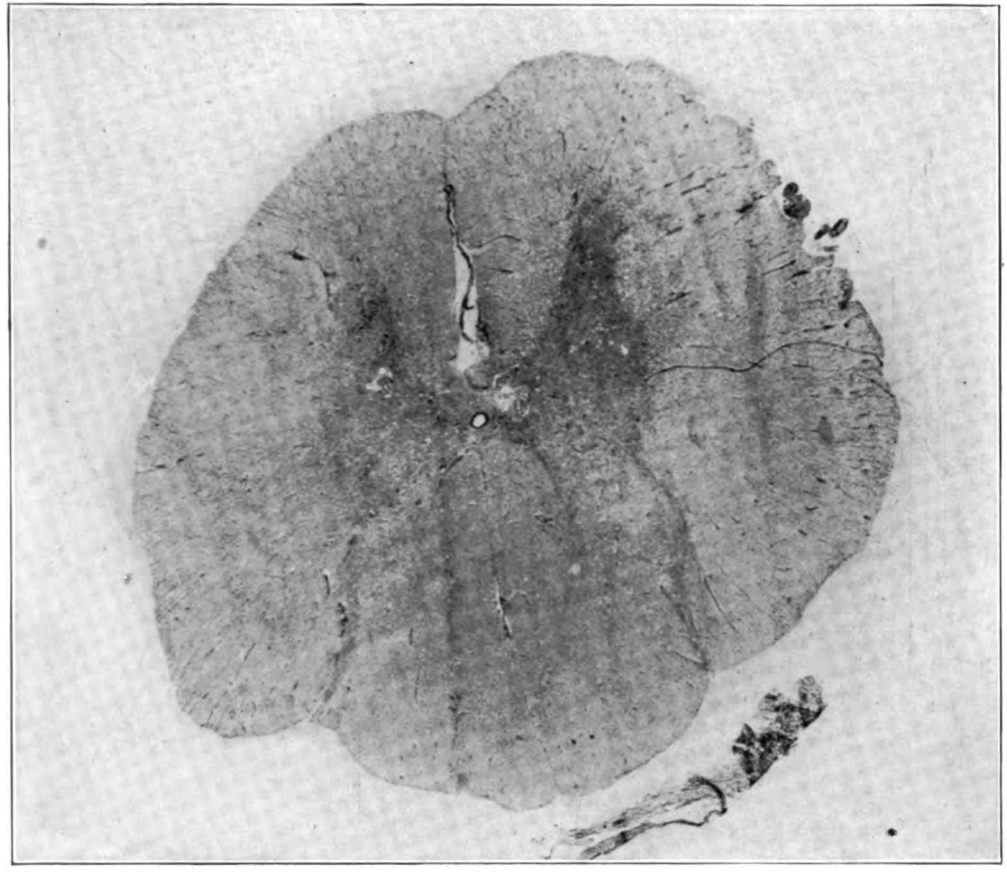

Fig. 1.—Cord (Case 1): anterior horns showing deep congestion, infiltration and edema.

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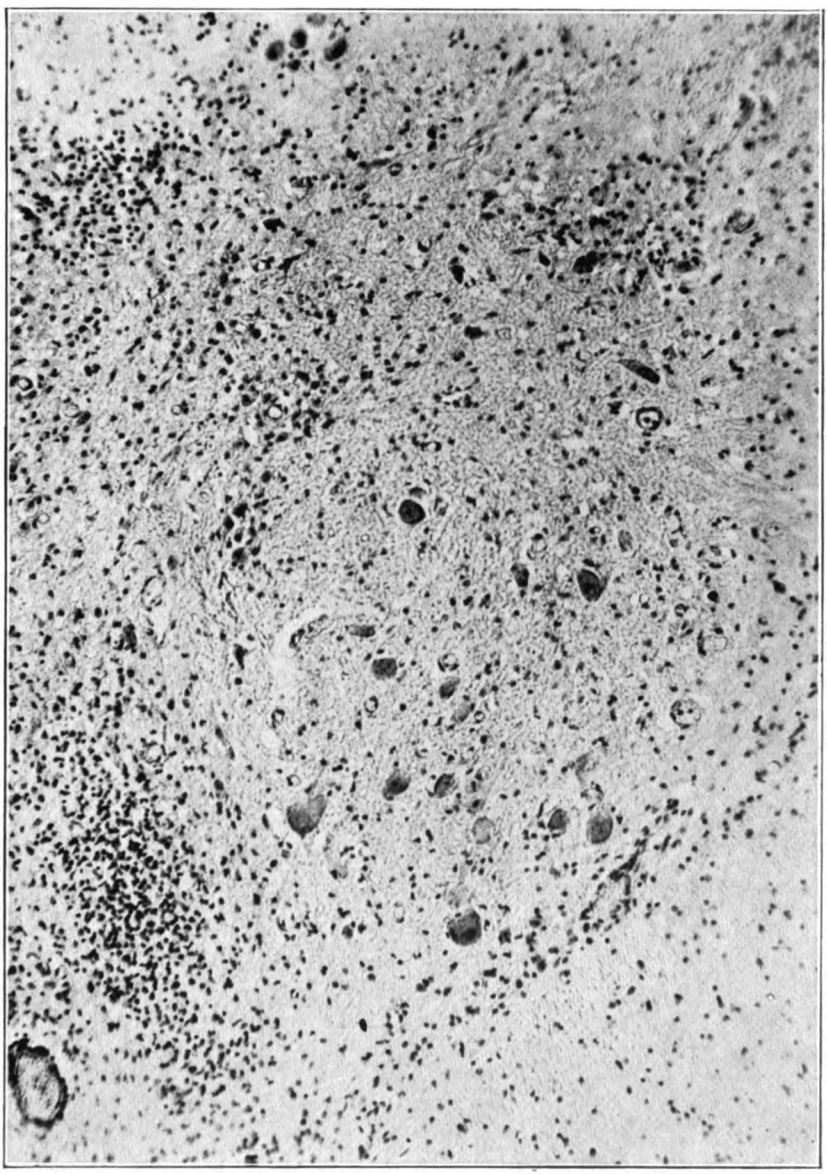

Fig. 2.—Cord (Case 1): a zone of infiltration surrounding a group of degenerating ganglion cells which show peripheral arrangement of Nissl's bodies.

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Fig. 3.—Cord (Case 2): hemorrhage and cellular infiltration in anterior horns.

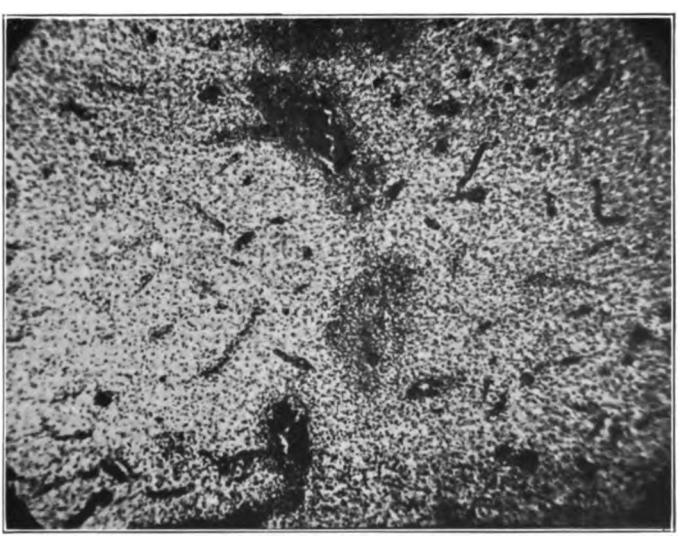

Fig. 4.—Enlarged view in anterior horns of cord shown in Figure 3.

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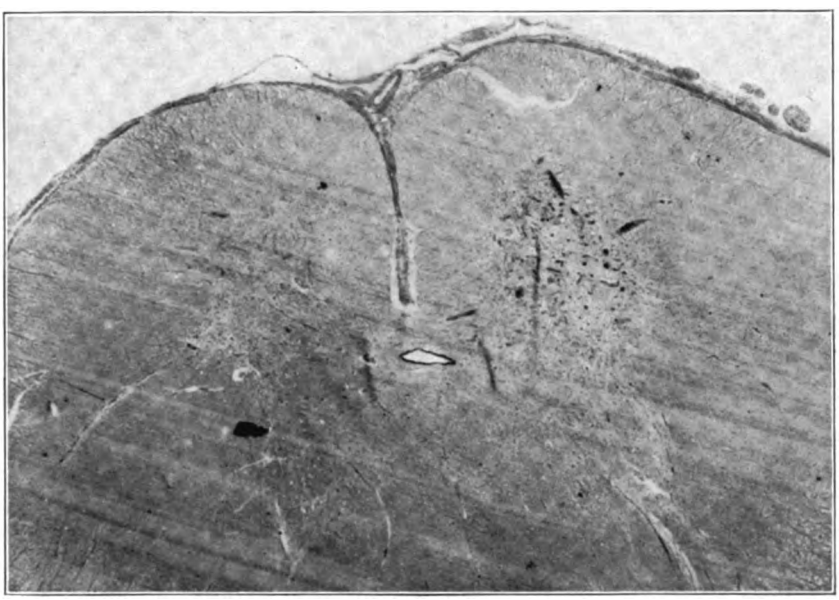

Fig. 5.—Cord (Case 3): hemorrhages and infiltration of anterior horns.

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Fig. 6.—Cord (Case 3), showing involvement of both anterior horns.

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Fig. 7.—Enlarged view of one side of cord shown in Figure 6 showing hemorrhage.

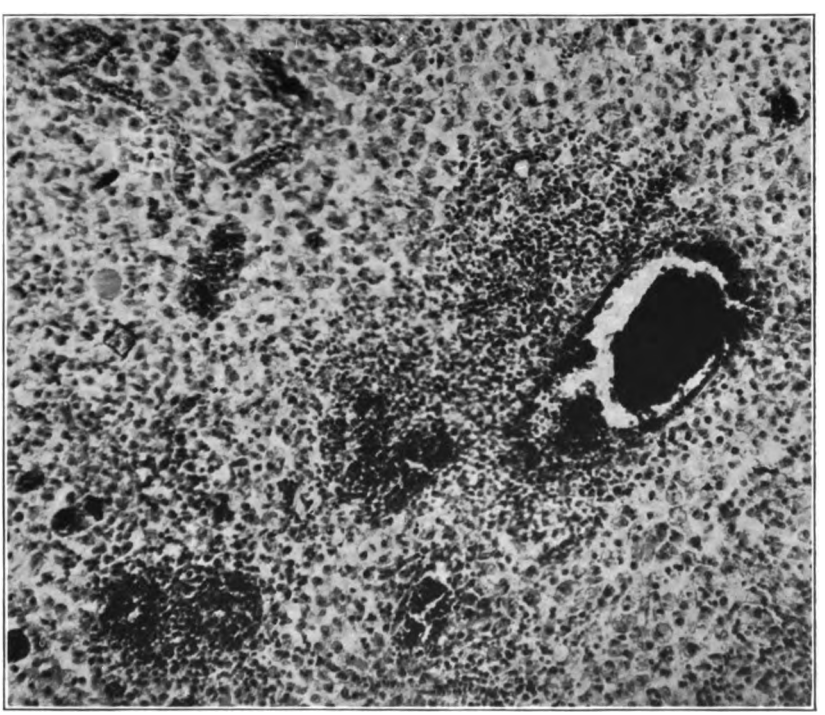

Fig. 8.—Area in Figure 7, showing hemorrhage in anterior horn with appearance of thrombosis.

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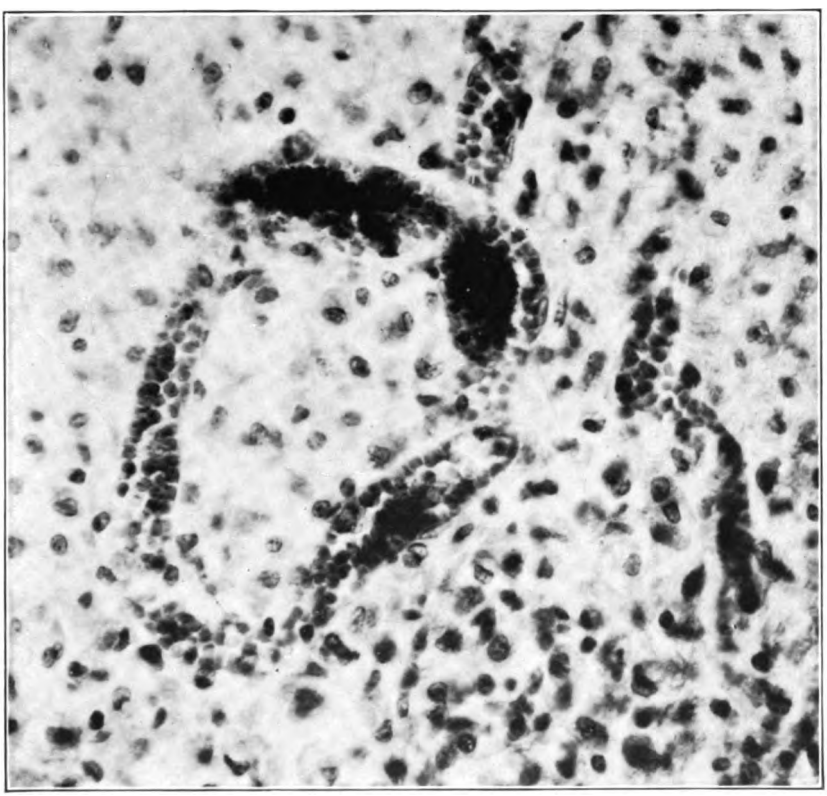

Fig. 9.—Area in Figure 7, showing dilated capillaries with stasis.

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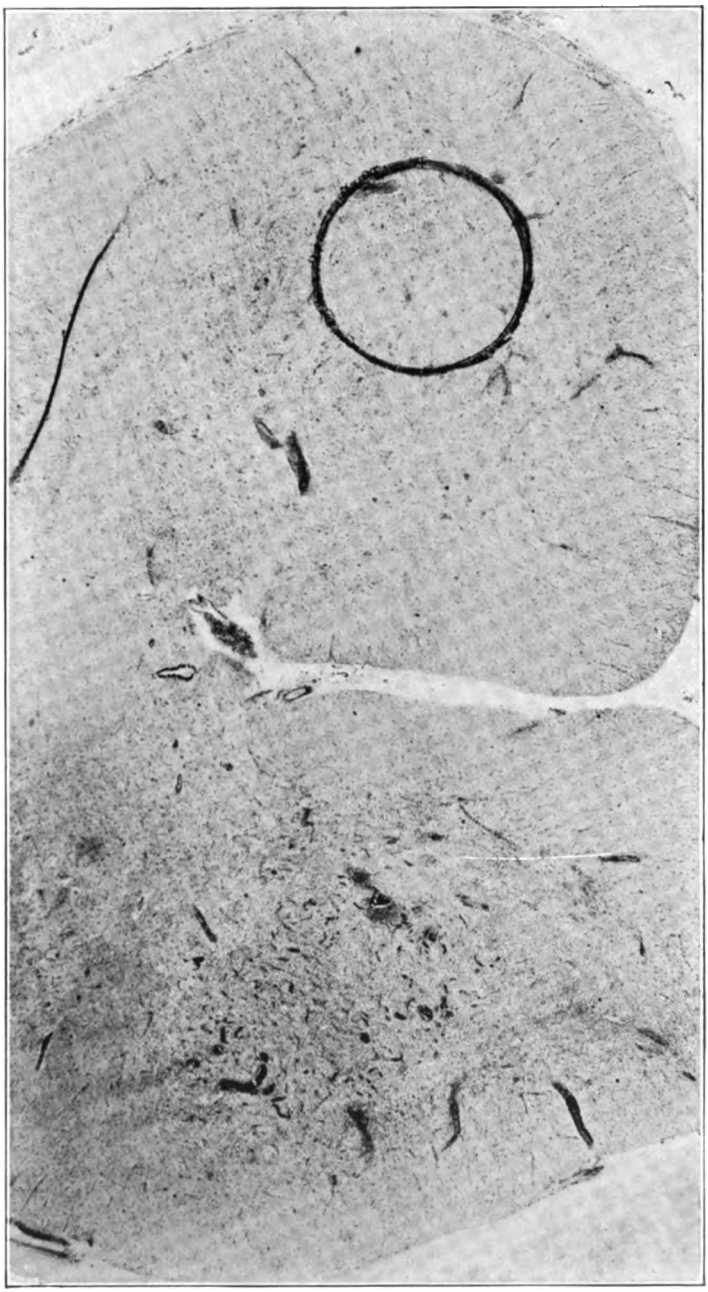

Fig. 10.—Cord (Case 3), showing hemorrhage into anterior horn of one side and perivascular groups of cells in white matter.

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normal. Kidneys congested, swollen and cloudy. Bladder and genital organs normal. Epiglottis, pharynx, larynx and trachea free from congestion or evidence of exudate. Thymus weighs 25 gm., is grayish pink in color and from cut surface a small amount of milky fluid escapes on pressure. Head normal to external examination. On removing calvarium and reflecting dura, vessels of pia-arachnoid are deeply congested. No increase of fluid in subarachnoid space. Brain removed with cord attached. Middle ears and bony air spaces normal. Careful section of brain shows no demonstrable lesion of cerebrum, lateral ventricles, cerebellum, basal ganglia or pons. Vessels of pia-arachnoid of cord deeply congested. No increase of spinal fluid, which is clear. Membranes free from exudate. In the lower portion of the medulla on the transversely cut surface is a softened area with a pin-head hemorrhagic center in the gray matter of the right side. Serial transverse sections of the cord, about 1 cm. apart, show numerous similar softened hemorrhagic areas in the gray matter of the anterior horns. In passing down the cord, they alternate fairly uniformly from one side to the other as far as the lumbar swelling, where hemorrhages appear on both sides. They average about 3 mm. in length and are most pronounced in the cervical and lumbar swellings. The cord shows swelling and softening of the affected sides.

Diagnoses.—Acute hemorrhagic anterior poliomyelitis. Congestion of pia-arachnoid of cord and brain. Congestion of spleen and lungs. Cloudy swelling of liver and kidneys. Partially patent foramen ovale.

Histologic Examination.[29]—Heart: Slight edema of connective tissue with swelling of muscle fibers. Lungs show dilated and congested vessels. Spleen: Blood-sinuses congested. Lymph-sinuses distended by lymphoid and proliferated endothelial cells. Malphigian bodies prominent and centers contain masses of endothelial cells, many of them phagocytic for other cells, nuclear fragments, and brown pigment. Liver: Edema of stroma and granular and vacuolar degeneration of pulp-cells. Sudan III stain in frozen sections shows extensive fatty changes. Colon normal. Kidney: Epithelial cells of tubules, especially the convoluted, swollen and lumina contain small amounts of precipitated serum. Capillary tufts of glomeruli and veins congested. Capsule and interstitial tissue edematous. Peribronchial lymph-node shows changes similar to those in the spleen, the phagocytosis being particularly marked. Thy-

29. Unless otherwise stated sections were prepared by imbedding in celloidin and staining with hematoxylin and eosin.

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mus: Hyperplasia of lymphoid elements. Dilated sinuses filled by lymphoid and enothelial cells; Hassall's corpuscles markedly degenerated, taking the eosin stain strongly and surrounded by a connective tissue sheath giving the appearance of lymph-sinuses thrombosed by masses of necrotic cells. Pituitary shows congestion of vessels of both lobes and dilatation of interlobar lymph-channels. Cerebrum and cerebellum, aside from congestion, especially of pial vessels, free from evidence of lesion.

Basal Ganglia: One vessel found with outer sheath containing polymorphonuclear leukocytes and lymphoid cells.

Medulla: In upper portion vessels uniformly congested. Perivascular lymph channels located beneath the ependymal surface of fourth ventricle are distended and packed with lymphoid and endothelial cells, the former in predominance. Many of the ganglion cells in this region are in various stages of degeneration, some showing peripheral arrangement of Nissl bodies with a clear perinuclear zone, others with faint staining nuclei and a swollen, granular, homogeneous cytoplasm, and still others showing loss of nucleus and beginning disruption of cell. Small foci of infiltrating cells appear in the nervous tissue, many of them polymorphonuclears and others resembling those seen around the vessels. In gray matter of lower portion is a focus of hemorrhage with destruction of nervous tissue and surrounded by a zone of edema with diffuse infiltration of cells.

Cervical Cord: The lymph-channels of the pia mater and those surrounding all vessels entering the cord are packed with lymphoid and endothelial cells, especially marked in that portion of the pia located in the anterior fissure, although the perivascular channels of the small branches entering the white matter are uniformly involved, especially those located near the ventral roots. Sections from various levels in this portion of the cord show the gray matter occupied by a hemorrhage, with surrounding zone densely infiltrated by inflammatory cells. In this region the ganglion cells are either degenerated or have disappeared entirely. The infiltration often extends backward into the gray matter of the posterior horn, involving Clarke's column. In the denser areas many nuclei are pycnotic and show varying stages of karyorrhexis, the nuclear fragments being sometimes taken up by the phagocytic cells. One particularly degenerated ganglion cell is found surrounded by a group of endothelial cells. The gray matter of the opposite side always shows more or less diffuse and focal infiltration but in many places, often in the

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region of infiltrated areas, apparently normal ganglion cells may be found. The capillaries in the gray matter are widely distended but show no evidence of thrombus formation. Occasionally the ventral nerve roots are edematous and diffusely infiltrated by cells of a lymphoid type. Cross-sections of many of the dilated vessels show apparently an increase in the number of lymphoid cells, suggesting a possible source of origin.

Thoracic Cord: Occasional small hemorrhages appear in the anterior horns but the cellular infiltration, even in the presence of the hemorrhages, is less marked than in the cervical portion.

Lumbar Cord: Hemorrhages appear in anterior horns of both sides with excessive destruction of the gray matter and very dense infiltration of cells. There is edema of the white matter extending into the anterior nerve-roots and even involving the posterior columns.

Sacral Cord: Free from hemorrhages, but shows both focal and diffuse infiltration of cells in the gray matter and packing of the perivascular spaces. Marchi stain at various levels failed to show definite evidence of degeneration. In sections stained by Gram-Weigert method the presence of micro-organisms could not be demonstrated.

Case 2.—Baby boy, aged 2½. Acute febrile attack with slight diarrhea. On the fifth day developed paralysis of right arm and leg, later involving left arm and leg. Death on eighth day from paralysis of respiratory muscles.

Autopsy.—July 29, 1909, eleven hours post-mortem. Body is that of a normally developed, well-nourished male infant. Body packed in ice and previously embalmed by injection of 2 oz. (?) of fluid in right axillary artery. Section limited to examination of spinal cord. On exposing dura of cord and incising region of cauda, about 30 c.c. of clear fluid escapes. On removing dura with enclosed cord and exposing pia-arachnoid, vessels are deeply congested. Surface clear and free from evidence of exudate. Odor of formaldehyd perceptible. Transverse sections made about 1 cm. apart. In upper portion of cervical enlargement in the anterior horn of the right side is a swollen, softened area showing a small hemorrhagic center. This hemorrhage measures about 3 mm. in length. No other gross hemorrhages observed, but the gray matter of both anterior horns in the cervical and lumbar swellings is softened and has a yellowish tinge.

Diagnosis.—Acute hemorrhagic anterior poliomyelitis.

Histologic Examination.—In sections from the cervical swelling one anterior horn is the seat of several small hemorrhages

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surrounded by dense masses of inflammatory cells, the endothelial type largely predominating. Many of these cells contain fragments of other cells, while others are swollen and their cytoplasm shows large vacuoles. Sections stained with thionin show many mitotic figures among these large cells, indicating active proliferation. The small capillaries are widely and often irregularly dilated and either closely packed with blood cells or empty, these latter giving the appearance of vessels previously over-distended, suddenly relieved of their contents and unable to return to normal caliber. This phenomenon never appears except in the region of hemorrhages. Ganglion cells in this area are wholly destroyed. The opposite horn is only slightly involved and shows many normal appearing nerve cells. The pial and perivascular changes resemble those seen in Case 1, except for the diminution in the number of polymorphonuclear leucocytes seen in the exudate. There is no evidence of thrombosis or embolism.

Sections from the thoracic and lumbar portions are free from hemorrhages but the gray matter of the anterior horns shows varying amounts of inflammatory infiltration and destruction of nerve elements, the lesions always being more pronounced on one side than on the other.

In the pial lymph-channels are found small groups of lymphoid and endothelial cells but in every case these are more numerous and more densely packed in the perivascular channels of the cord substance. The lumbar region is not as markedly involved as the cervical. As in Case 1, the infiltration often extends into the gray matter of the posterior horns, and the adventitial sheaths of the vessels of the posterior columns occasionally contain groups of cells.

Gram-Weigert stain for organisms and special stains for demonstrating the presence of Negri or other intracellular bodies in the ganglion cells were negative.

Case 3.—Baby girl, aged 3. Acute attack of high fever, accompanied by aching of lower limbs and pain in back. Motor paralysis of both legs and left arm noticed on third day. On the following day there was complete paralysis of all extremities and beginning involvement of the muscles of respiration. Death on twelfth day.

Autopsy.—Oct. 8, 1909, two and one-half hours post-mortem. Body is that of a normally developed, well-nourished female infant. The skin of face, neck, trunk and upper portions of limbs is thickly studded with a white miliary papulo-vesicular eruption. (This had followed the injection of a dose of anti-streptococcic serum during the illness.) Peritoneal, pleural

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and pericardial cavities normal. Beneath epicardium are a few petechial hemorrhages along the line of the left coronary artery. Heart muscle shows cloudy swelling. Lungs contain dark congested areas in dependent portions. No evidence of nodules or consolidation. Mucous membrane of bronchi reddened and lumina filled with frothy mucus. Peribronchial lymph nodes swollen and congested. Spleen normal in size. Pulp congested, soft, and lymph nodules are prominent. Liver is swollen and cloudy. Pancreas and gastro-intestinal tract and adrenals normal. Kidneys swollen, deeply congested and cut surfaces cloudy. Pharynx, tonsils, larynx, trachea, thyroid and thymus free from any evidence of inflammation or disease.

Head: On removing calvarium and reflecting dura, the vessels of pia-arachnoid are deeply injected. Careful sectioning of brain, including pons and medulla, fails to reveal gross lesion.

Cord: Examination of cord shows deep congestion of vessels. Cerebrospinal fluid is clear and not increased in amount. No evidence of exudate on surface. Sectioned transversely, upper cervical region shows congestion of the anterior horns. In the cervical swelling a softened hemorrhagic area involves the entire left anterior horn. Below the cervical swelling a small hemorrhagic focus appears in the right anterior horn. The thoracic portion, aside from congestion, especially of the gray matter, appears normal for a distance of 6 cm., when a small hemorrhage again appears on the left side. In the lumbar swelling hemorrhages are present on both sides and involve the gray matter of the anterior horns as far as the last sacral segment.

Diagnoses.—Acute hemorrhagic anterior poliomyelitis. Congestion of pia-arachnoid of brain and cord. Cloudy swelling of heart, liver and kidneys. Subepicardial hemorrhages. Papulo-vesicular cutaneous eruption.

Histologic Examination.—Sections of the abdominal and thoracic viscera show conditions closely resembling those found in Case 1, the changes in the spleen, lymph-nodes, and thymus being fully as prominent as those previously described. Cerebrum and cerebellum free from evidence of lesion except for deep congestion of vessels and the presence of occasional polymorphonuclear leukocytes and lymphoid cells in the tissue spaces of the pia-arachnoid. Cross-sections of the blood-vessels of the pia show an apparent increase in the total number of polymorphonuclear leukocytes. Sections of basal ganglia and cerebral peduncles show more intense congestion of vessels and occasional groups of cells in the surrounding lymph-

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channels of the small branches. This infiltration around the vessels becomes more marked in the upper portion of the medulla, and near the lower portion beneath the ependyma of the fourth ventricle a small hemorrhage appears, with extensive diffuse infiltration of lymphoid and endothelial cells, accompanied by edema of nervous tissue. The ganglion cells in this region are in various stages of degeneration, some being wholly necrotic. The perivascular spaces are packed with cells and near the affected areas the red blood corpuscles are densely massed in the irregularly distended capillaries, so that often, even in very thin sections, they appear fused into a more or less homogeneous mass, resembling a forming thrombus. In passing down the cord, sections at various levels show areas of hemorrhage and inflammatory infiltration similar to those observed in Cases 1 and 2, but much more intense in degree. These areas are centered always in the gray matter of the anterior horns, but, as before noted, often invade the posterior portions of the cord. Near the foci of hemorrhage in every instance the dilated capillaries suggest the appearance of complete stasis of the blood stream. The lymph channels of the pia mater throughout the entire cord are noticeably less infiltrated than those surrounding the vessels of the cord itself. Cross sections of vessels show increase in the number of polymorphonuclear leukocytes, but very few cells of this type appear in the exudate. Sections of cord with nerve roots and also of nerves of cauda stained by Marchi method present occasional fibers with beginning degeneration.

Case 4.—A girl, aged 6, suffered typical acute attack in the early part of August, 1909, and rapidly developed complete motor paralysis of both lower limbs and partial paralysis of upper limbs, more marked on the left side.[30] October 13 she was admitted to the State Hospital for Crippled and Deformed Children and on October 22 developed an attack of broncho-pneumonia. By November 1 she had apparently completely recovered from this. December 25 was seized with an attack of labored breathing; a few hours later showed clear evidences of pneumonia. Death occurred on Jan. 3, 1910, nearly five months after the acute attack.

Autopsy.—Jan. 5, 1910, fifty hours post-mortem. Body is that of a fairly well-developed, poorly nourished female child. Muscles of both upper and lower extremities show decided

30. A 2-year-old boy and a month-old baby in this family were taken sick at the same time and died after a few days' illness. The clinical diagnosis was anterior poliomyelitis in both cases; no autopsies obtained.

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atrophy, those of the right arm and forearm being least affected. The lungs show patches of consolidation almost uniformly scattered over all the lobes of both sides. From cut bronchioles purulent fluid escapes on pressure. Peribronchial lymph nodes soft and swollen. Remaining viscera of trunk, aside from deep congestion and cloudy swelling of liver and kidneys, are apparently normal. Head not examined. On incising dura of cord, in the lower portion of spinal canal a normal amount of clear serum escapes. On removing cord and making transverse incisions about 1 cm. apart the following lesions appear: In the upper portion of the cervical swelling in the anterior horn of the left side is a softened, depressed, grayish-red area. This lesion extends through the cervical swelling. About the mid-portion a similar but smaller area appears on the right side. In the midthoracic portion as far down as the lumbar swelling, minute, reddish, softened areas are seen at intervals in the anterior horns. In the lumbar swelling both anterior horns are softened and of a grayish and yellowish-red color. The sacral cord is apparently more nearly normal. Portions of mid-brain removed through foramen magnum.

Diagnoses.—Acute bronchopneumonia. Anterior poliomyelitis with softening of cord and atrophy of muscles of limbs.

Histologic Examination.—Heart: Swelling of muscle fibers and edema of interstitial tissue. Lungs: Well-developed bronchopneumonia showing, in addition to the usual features, marked hyperplasia of the peribronchial lymphoid tissue. Lymph-node (mediastinal): Marked dilatation of sinuses which are filled with lymphoid, polymorphonuclear and proliferated endothelial cells, many of which are phagocytic for other cells and brown pigment granules. Spleen: Sinuses resemble those of lymph-node. Centers of Malpighian bodies filled with masses of endothelial-like cells undergoing hyaline fibrinoid changes. Liver and kidneys show congestion, granular degeneration of the pulp cells, and edema of connective tissue. Thymus resembles organ in Case 1, except for atrophy of lymphoid elements and increase of fat-bearing connective tissue.

Mid-brain: A section passing through the substantia nigra shows one large vessel with its outer sheath closely packed by lymphoid cells. The characteristically pigmented ganglion cells are in the early stages of degeneration.

Spinal Cord: The location and extent of the lesions at various levels differ very little from those described in the previous cases, but the appearances of these lesions present

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the following differences: 1. The pial and perivascular infiltration is less dense and composed almost wholly of lymphoid cells. 2. The gray matter of the affected anterior horns shows wide edematous spaces with fewer cells. 3. These cells are uniformly swollen with small dark-staining nuclei pushed to one side. Staining by the Marchi method demonstrates varying sized fat globules in the cytoplasm. Occasional lymphoid cells appear in the loose meshes. 4. In these areas a few ganglion cells remain. These are shrunken and their cytoplasm often densely packed by small basic-staining granules. Sometimes such a cell is surrounded by a large number of oval vesicular nuclei giving the appearance of a foreign body giant cell (neuronophage). Small multinucleated cells are fairly common. 5. In these same regions blood vessels are noticeably reduced in size and number and widely separated by large edematous spaces. The Marchi stain reveals advanced fatty changes in the walls of these vessels. 6. In portions of the thoracic cord there is shrinking of the gray matter of the affected side but nowhere is there any evidence of proliferation of neuroglia tissue or vascular repair. 7. Sections of the anterior nerve-roots and bundles of nerves from cauda and sacral plexus show varying degrees of degeneration when stained by the Marchi method.

Case 5. Girl, aged 15. Acute attack of chills and fever lasting two days, followed by motor paralysis of lower extremities and rapid movement of upper extremities except fingers of right hand. On the eleventh day she had a second chill, followed by fever and labored breathing. Death occurred on fifteenth day.

Autopsy.—Dec. 12, 1909, thirteen hours post-mortem. Body is that of a well-developed, poorly nourished young girl. Serous cavities and heart normal. The left lung shows an area in each lobe posteriorly which is dark bluish-red in color, non-crepitant and firm. Cut surface is rather dry and granular. In the dependent portions of right lower lobe are numerous scattered rounded nodules of consolidation. The mucous membrane of the bronchi is injected and the lumina contain frothy mucopurulent fluid. Peribronchial lymph-nodes swollen and congested. One shows a small focus of calcification. Liver, spleen and kidneys apparently normal except for congestion. Thymus atrophied. Brain surface deeply congested. No excess of fluid. Careful section shows no demonstrable lesion in cerebrum, cerebellum, or basal ganglia. On the left side of the medulla is a pin-point hemorrhage located in the gray matter. Middle ears and bony air

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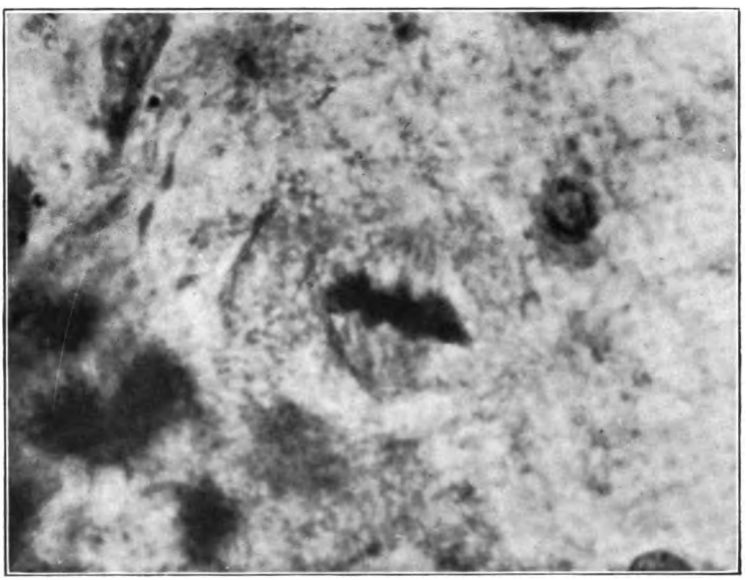

Fig. 11.—Endothelial cells in mitosis, found within circle of Figure 10.

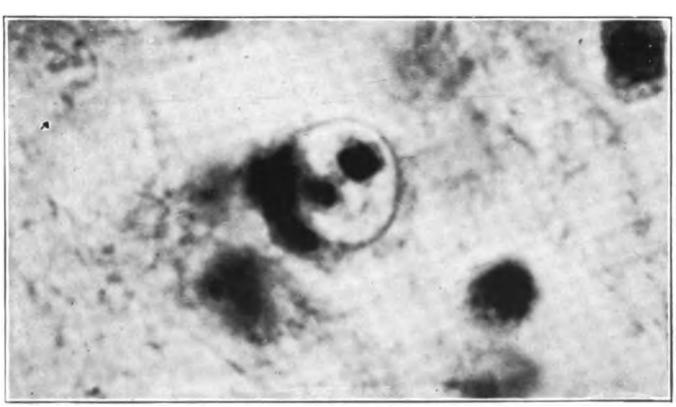

Fig. 12.—Endothelial cells inclosing nuclear fragments, found within circle of Figure 10.

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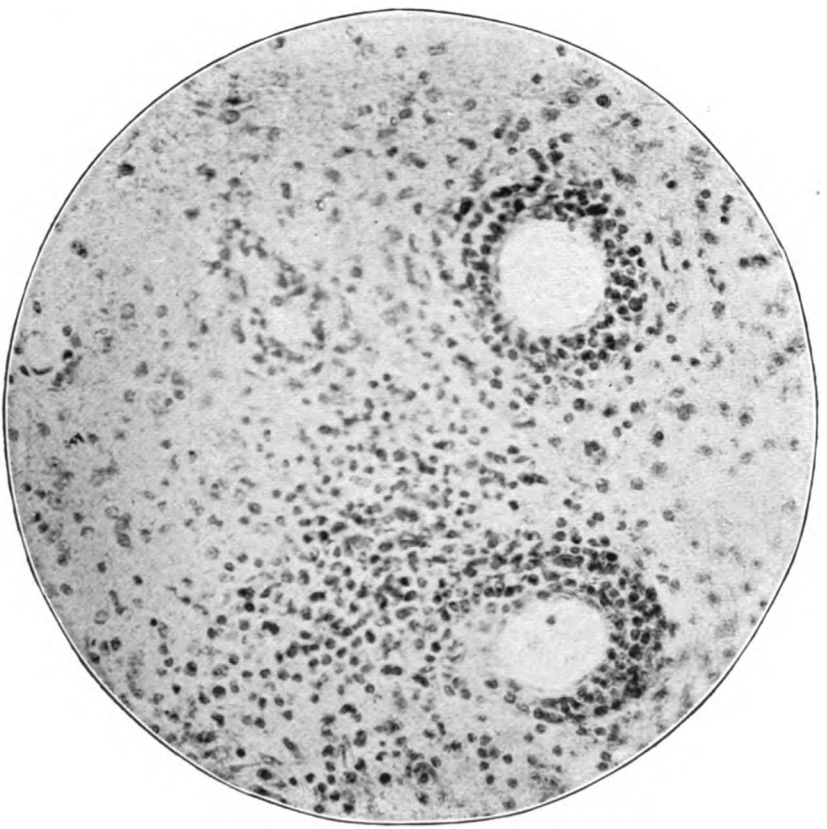

Fig. 13.—Perivascular collection of cells with beginning diffuse infiltration (Case 3).

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Fig. 14.—Anterior horns of cord (Case 4), showing advanced destruction of gray matter.

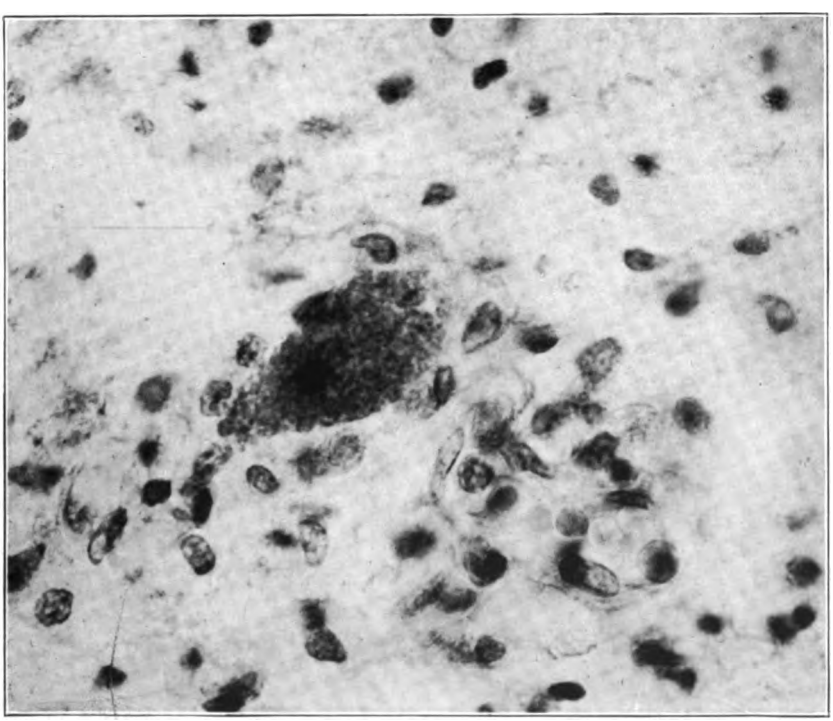

Fig. 15.—Degenerating ganglion cell with beginning giant cell formation (Case 4).

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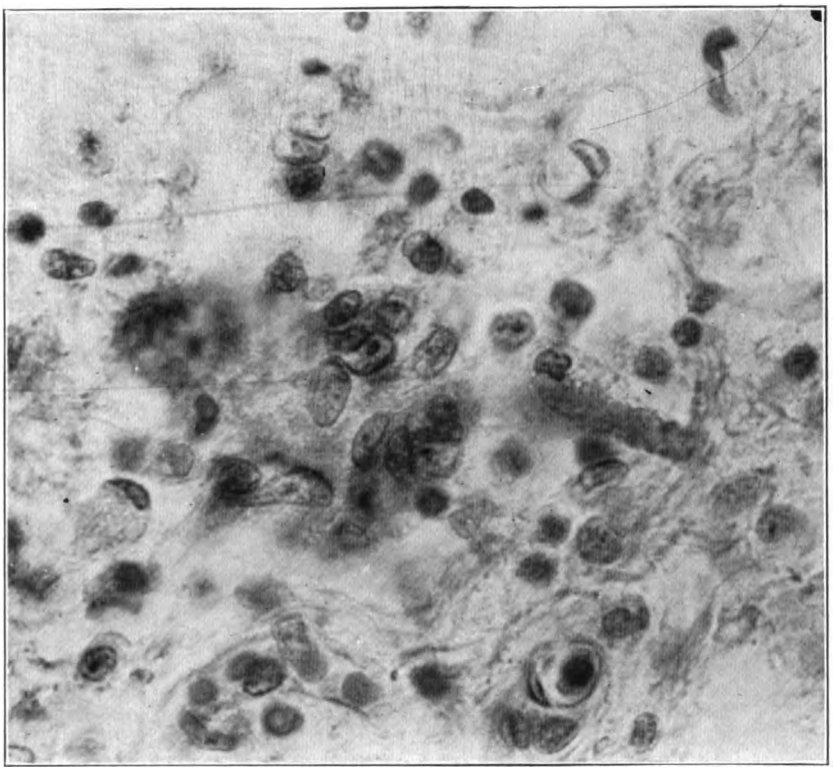

Fig. 16.—Giant-cell inclosing remains of ganglion cell (Case 4).

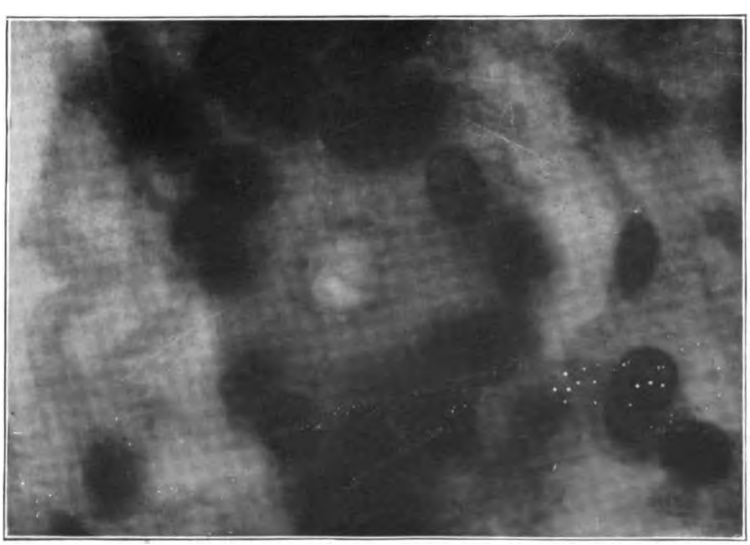

Fig. 17.—Giant-cell formation (Case 14).

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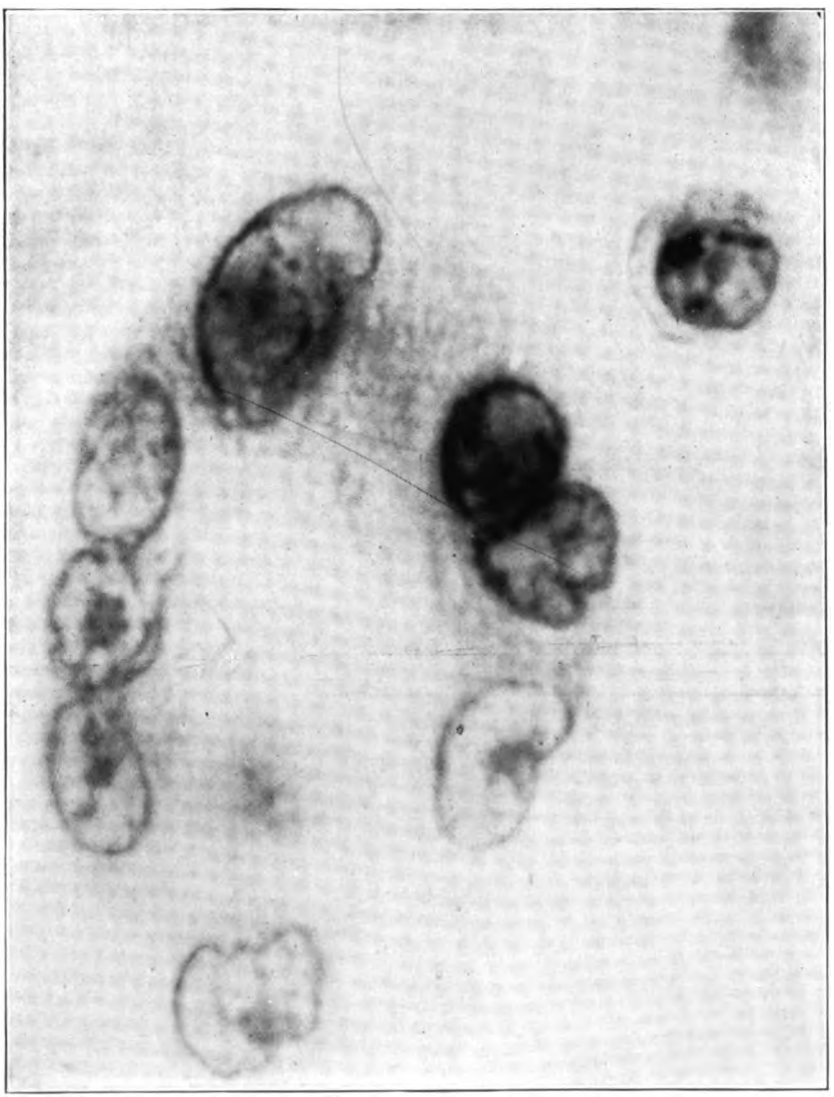

Fig. 18.—Large multinucleated cell (Case 4).

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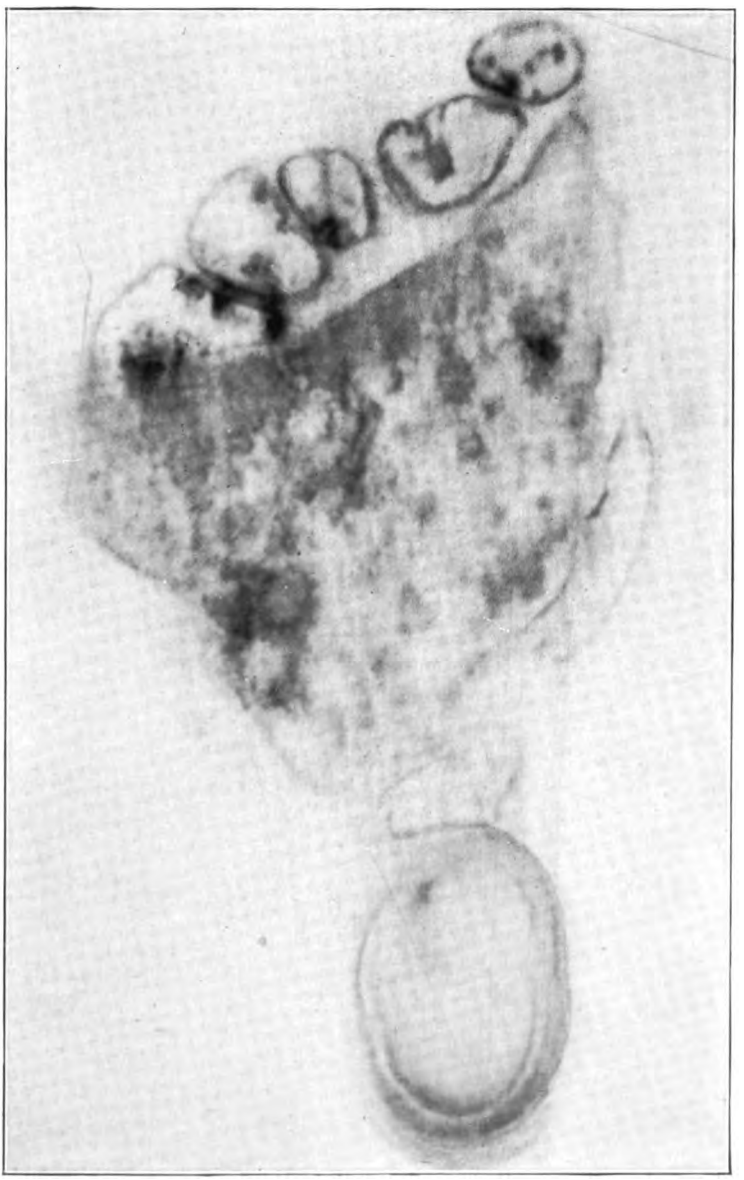

Fig. 19.—Giant cells enclosing nerve cell with adjacent foreign body (Case 4).

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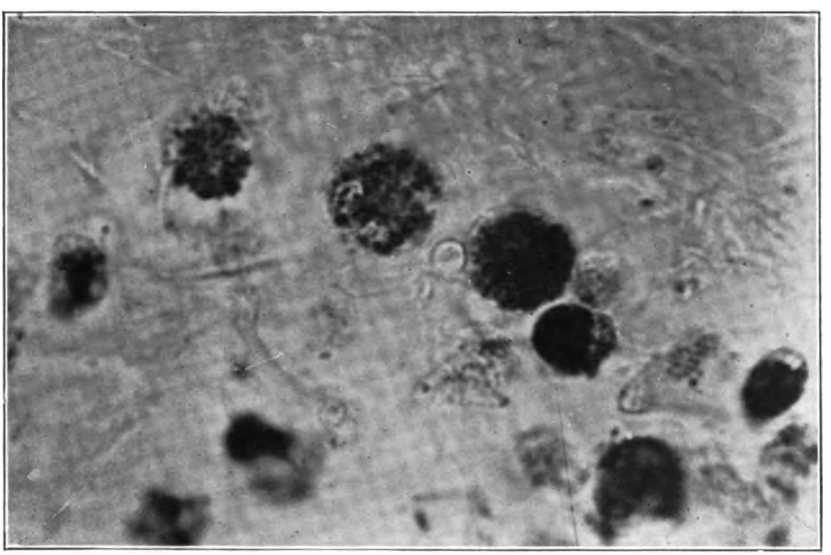

Fig. 20.—Endothelial cells in gray matter containing fat droplets (Case 4), osmic acid stain.

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Fig. 21.—Fatty degeneration of cells of blood-vessel walls (Case 4); osmic acid stain.

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spaces normal. Cord shows congested membranes with only a small amount of clear spinal fluid. In the cervical cord the gray matter of the anterior horns is reddened and softened. In the cervical swelling hemorrhages appear on both sides. The thoracic portion is free from hemorrhages but the gray matter is soft, congested and swollen. Both anterior horns in the lumbar enlargement show hemorrhages and swelling of the cord.

Diagnoses.—Acute hemorrhagic anterior poliomyelitis. Acute terminal broncho-pneumonia. Congestion of membranes of cord and brain. Calcification of bronchial lymph node.

Histologic Examination.—Lungs: Sections from dependent portions give the typical appearances of broncho-pneumonia. Bronchial lymph-node resembles that described in Case 4. Aside from congestion, liver, kidneys and spleen appear normal. Lymphoid tissue of thymus gland reduced in amount and Hassall's corpuscles are very prominent, suggesting the appearances seen in the thymus in Case 1.

Medulla: In the gray matter of one side is a focus of hemorrhage surrounded by a very dense mass of inflammatory cells. In this area the ganglion cells are uniformly necrotic, appearing as swollen, eosin-staining, homogeneous bodies. In the cells of the exudate karyorrhexis is prominent, evidenced by innumerable basic-staining granules of nuclear chromatin. These granules are often contained in the cytoplasm of the endothelial cells which are numerous both in this exudate and in that surrounding the blood vessels. The opposite side shows a slightly lessened amount of infiltration without hemorrhage, but only an occasional ganglion cell may be found. The blood-vessels are dilated and their contents sometimes simulate the appearance of thrombosis, as in Case 3. Sections from lower levels differ little from those described in Case 1. The absence of polymorphonuclear leukocytes from the exudate is a marked feature. The anterior nerve-roots show a slight lymphoid cell infiltration and their blood-vessels, as well as those of the posterior roots, are widely dilated. In sections of the spinal ganglia opposite the lumbar swelling there is degeneration and swelling of the ganglion-cells and edema with lymphoid cell infiltration of the interstitial tissue. Marchi stain demonstrates advanced fatty changes in the nerve fibers.

Case 6.—Boy, aged 17. Taken sick with dull pains in chest and back and general prostration. Two days later had a severe chill followed by high fever accompanied by pain in chest, cough, and vomiting. No history of paralysis could be

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obtained, but the father stated that shortly before death patient developed difficulty in swallowing. Death on fourth day.

Autopsy.—Oct. 16, 1909, thirty-three hours post mortem. Body is that of a normally developed, poorly nourished boy showing slight general subcutaneous edema. Mesenteric lymph-bodies markedly swollen, especially near the cecum where the largest is bean-shaped and fully 4 cm. in length. Pleural cavities irregularly interrupted by tough fibrous adhesions. Heart shows numerous fine hemorrhages beneath epicardium; muscle soft and cloudy. Lungs show similiar subpleural hemorrhages. Both lower lobes are thickly studded with small patches and nodules of consolidation. On section of these areas, from cut bronchioles thick, yellow pus escapes on pressure. In lingula of left lung are two large, wedge-shaped areas of interstitial emphysema. Left apex shows a firm fibrous nodule containing a focus of calcification. Lymph-nodes at bifurcation of trachea contain caseous centers with calcareous capsules. Spleen swollen and soft. Pulp scrapes easily and lymph nodules are prominent. Peyer's patches and solitary follicles in ileum are raised, swollen and deeply congested. Kidneys large, pale and soft. Cut surfaces cloudy. Urine removed post mortem shows large amount of albumin and various forms of casts. Aorta presents numerous fine yellowish streaks of atheroma. Trachea congested but shows no evidence of membrane formation. Mucous membrane of throat dark and swollen. Tonsils swollen. No evidence of diphtheria. Head not examined. Cord shows congestion of membranes. Cerebrospinal fluid clear and not excessive in amount. Serial transverse sections of cord show no gross evidence of lesion beyond a slight reddening of gray matter of anterior horns.

Diagnoses.—Acute broncho-pneumonia. Acute diffuse nephritis. General lymphoid hyperplasia. Old healed pulmonary tuberculosis. Old healed pleuritis.

Histologic Examination.—Heart: Muscle cells swollen and striations indistinct. Lung: Typical patches of broncho-pneumonia with peribronchial lymphoid hyperplasia. Lymph-nodes (bronchial and mesenteric): Extreme congestion; otherwise resemble those of Case 4. Spleen: Congestion marked. Centers of lymph nodules packed with endothelial cells. Intestines: Lymphoid structures are hyperplastic and central portions of follicles and sinuses resemble those of spleen and lymph-nodes. Liver: Extreme congestion, especially in central zones of lobules. Granular and vacuolar changes in pulp cells. Adrenal: Congestion of considerable duration evidenced by

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atrophy of cell cords of inner zone. Kidney: Acute diffuse nephritis of the tubular type, such as often found in acute toxic fevers of the bacterial origin.

Cord: Sections at various levels free from any evidence of hemorrhages. In the lymph-spaces of the pia, especially that portion located in the anterior fissure, there is abundant exudate of cells which are largely of the polymorphonuclear type. A few lymphoid and endothelial cells appear, many of the latter showing phagocytosis. The perivascular spaces in the anterior portions of the cord are closely packed with a similar exudate and in the gray matter are numerous foci of cells composed chiefly of polymorphonuclear leukocytes. Some of the ganglion cells in these regions are in various stages of degeneration but they are not apparently diminished in number and many normal appearing cells may be seen. No large or even small areas of gross destruction of gray matter, such as described in the other cases, can be found. The vessels are congested but show no evidence of thrombosis or even advanced stasis. The cervical and lumbar swellings are most markedly affected and one anterior horn is always more involved than the other. The posterior horns and columns are uniformly unaffected.

SUMMARY

Cases 1, 2, and 3 represent the ordinary type of acute lesion with hemorrhages. Cases 4, 5 and 6 are complicated by pneumonia. In case 4 the patient probably would have died eventually; the pneumonia being only a terminal affair. Case 5 was an institutional case, and the patient, though safely through the acute stages, succumbed to intercurrent infection. Case 6 was so complicated by the occurrence of pneumonia and acute diffuse nephritis that we are inclined to believe the attack of poliomyelitis was only a minor event; and pathologically the lesion certainly is less severe, or more early, than that presented by any of the other cases.

The process as a whole follows a definite and specific course. Whatever may be finally shown to be the agent of infection its "optimus locus" is in the perivascular lymph-channels of the anterior portions of the cord, especially those located in the gray matter. From this site the reaction spreads, on the one hand, to the sur-

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rounding tissue spaces of the gray matter, and on the other, to the lymph-channels of the white matter and pia, often involving the posterior portions of the cord. We are inclined to believe these channels also represent the avenue of infection, whether it starts from the nasal or some other external surface.

The cells collecting in these spaces are not those to be expected from an ordinary pyogenic infection. Very early in the disease polymorphonuclears appear in small numbers, but they are soon replaced by endothelial cells and lymphocytes, the latter predominating by the end of the second week. The small number of the polymorphonuclear leukocytes in the exudate of all but one of the cases was a noticeable feature. The cellular reaction alone somewhat resembles that seen in the primary and secondary lesions of syphilis. The lymphocytes apparently come from the blood and lymph-streams combined. In cross-sections of dilated vessels their number is often increased and at one point in a vessel wall active migration of these cells was observed. The endothelial cells arise, we believe, from proliferation of those normally lining the lymph-channels and spaces. In several areas they showed mitotic figures. They are phagocytic for whole cells and disrupted portions of cells and in turn undergo fatty degeneration of cytoplasm and disintegration. They appear in especially large numbers in the diffuse exudate in the anterior gray matter.

The toxic action of the disease is chiefly directed against the anterior horns. The first noticeable change in addition to the congestion and perivascular collection of cells is edema in the interstitial spaces. This is rapidly followed by focal or diffuse infiltration of cells with or without hemorrhages and accompanied by destruction of both the neuroglia and ganglion cells. Many of these latter, however, persist and sometimes appear normal even in the region of extensive infiltration. They may show various stages of degeneration from irregular grouping and peripheral arrangement of Nissl's

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granules to edema of cells, loss of nucleus and complete disintegration. In the most prolonged case of this series (Case 5), nerve cells were found with shrunken outlines and cytoplasm crowded with large basic-staining granules. One of these cells was surrounded by a large number of oval vesicular nuclei, suggesting a giant-cell or so-called neuronophage. In the later stages the hemorrhage is absorbed, the cells of the exudate disintegrate and proliferation of neuroglia begins the process of repair. The blood-vessels are always congested, especially the capillaries and veins. The Marchi stain demonstrates fatty degeneration in their walls and this factor probably accounts for the loss of elasticity, irregular dilatation and final rupture. We could not satisfy ourselves of the presence of thrombosis in any of our cases. The question does not seem an important one. Granted that the vessel-walls are diseased and overdistended and that the blood-current is so obstructed that it approaches a condition of stasis, then hemorrhages may occur, whether actual thrombus formation is or is not present. These hemorrhages are the most important factor in producing extensive destruction, occurring as they do almost uniformly in the gray matter of the anterior horns. More than any other one lesion they probably tend to bring about a fatal result.

Staining by the Marchi method demonstrates patchy degeneration of nerves in both anterior roots and peripheral nerve fibers. This occurs fairly early in the disease. Special stains for micro-organisms, such as Gram-Weigert, eosin and methylene blue and thionin, were uniformly negative.

Why the process spends itself on the anterior horns has always been a question for active discussion. Undoubtedly the blood-supply is especially abundant through the anterior spinal arteries, but abundant blood-supply in other portions of the body is not usually considered the most important determining factor for the location of disease processes. That the lesion is specific

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and definitely selective seems by far the most reasonable supposition and one that conforms with numerous analogies in both the cord and other portions of the body, such as tabes dorsalis, typhoid fever, etc.

The location of the lesions has been described under each case. The medulla and cervical and lumbar swellings were most often the seat of hemorrhages, indicating that at these points the selective action of the virus was most manifest. We were not able to find evidence of involvement of the cerebrum or cerebellum but in two cases the cerebral peduncles and in one instance the basal nuclei showed vessels surrounded by collections of cells.

One noticeable feature not sufficiently emphasized in the literature is the evidence of a general toxemia. The parenchyma of the heart, liver and kidneys was the seat of cloudy swelling and invariably the lymph-nodes and spleen showed the customary reaction to the presence of a toxin in the general circulation. Hyperplasia of lymphoid tissue with proliferated and often phagocytic endothelial cells collecting in the sinuses and contral portions of the follicles was constantly observed.

Through the kindness of Drs. Flexner and Lewis, we were able to compare the lesions found in our cases with those obtained in the spinal cords of monkeys experimentally inoculated with the poliomyelitis virus. Perivascular collections of cells, diffuse infiltration and hemorrhages in the anterior gray matter were present in various stages. While the structure of the monkey's cord differs somewhat from the human, the lesions seem in every way analogous to those found in our cases.

CONCLUSIONS

1. Acute anterior poliomyelitis is a specific infectious disease characterized pathologically by general toxemia affecting the parenchyma of the heart, liver and kidneys and the lymphoid tissues of the body, but spending itself locally on the structures of the spinal cord.

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2. Grossly the cord is congested and on transverse section shows softening and often hemorrhages in the gray matter of the anterior horns.

3. In the cord the infectious agent is located in the perivascular lymph-channels of the anterior portions, especially invading the gray matter, but extending to the white matter and pia and occasionally the posterior horns. The brain stem and basal ganglia may be involved. In the cord the medulla and cervical and lumbar swellings are particularly affected.

4. The characteristic lesion consists of collections of cells in the perivascular and pial lymph-channels and tissue spaces of the anterior horns. Of these cells the polymorphonuclear leukocytes appear early and are relatively few in number. They are soon displaced by endothelial cells arising from proliferation of the lining endothelium and lymphocytes coming from the blood and lymph streams.

5. Edema of the interstitial tissue and degeneration and destruction of the ganglion cells are always present.

6. The vessels are congested, their walls degenerated, and the capillary branches in the gray matter irregularly distended and often ruptured, giving hemorrhages, which always intensify markedly the amount of destruction. Thrombosis was not observed.

7. Early degeneration of nerve fibers from the anterior roots is a constant feature.

8. Stains for micro-organisms were uniformly negative.

II. BACTERIOLOGY

By A. J. Chesley, M.D.

To attain our present conception of poliomyelitis the symptomatology and pathology have been broadened to include many variations in symptoms and lesions. To determine the cause of poliomyelitis the etiology has been narrowed to exclude many factors once classified as exciting or causing the disease. All the common diseases, most of the accidents and tribulations incident

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to childhood have been so classified. Having no evidence of hereditary transmission, and being unable to accept the maternal impression theory, conservative writers advanced a theory that the disease might be due to infection with a specific micro-organism.

A review of the literature shows a remarkable variety and inconstancy in the bacteriologic findings of spinal fluids and autopsy materials. At best, only temporary credence has been given the belief in any micro-organism as the probable cause of the disease. For example, the following list is given, which includes a sufficient number of the infectious agents mentioned as probable causes: the staphylococcus (all varieties), the streptococcus, meningococcus, pneumococcus, *Micrococcus catarrhalis*; the bacillus of diphtheria, of anthrax, of influenza, of typhoid, the colon bacillus, the malarial parasite; the virus of rabies, of smallpox, of measles, of scarlet fever, of syphilis, and of whooping-cough.

A Gram-positive coccus, now generally known as Geirsvold's diplococcus (in appreciation of Geirsvold's important work during the Scandinavian epidemic) has received the most attention. Geirswold's etiologic theory, expressed in very conservative terms, brought this coccus into serious consideration. It has been found in purity in many specimens of spinal fluid withdrawn during life, and in the organs of several patients after death. It has produced symptoms closely resembling those of human poliomyelitis in a few of the animals experimentally infected. It has been recovered in purity from the spinal fluid and tissues of such animals. Some of this work has been done with great care to eliminate the possibility of contamination, and control experiments have practically excluded this possibilty. The crucial test is the search for the specific lesions of poliomyelitis. When lesions have been present in the spinal cords of rabbits, guinea-pigs, mice, rats, pigeons or monkeys inoculated with living cultures, or spinal fluid from which pure cultures of Geirsvold's diplococcus were ob-

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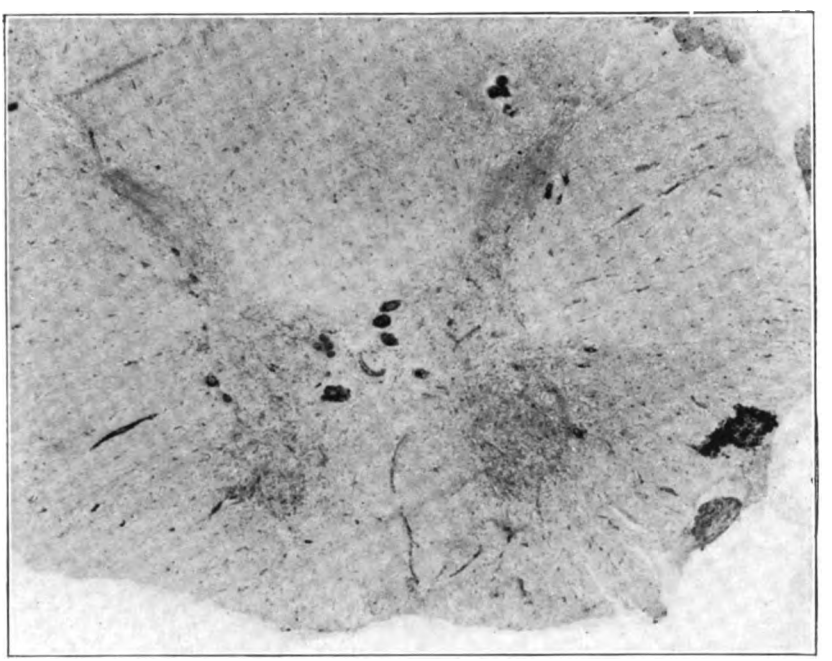

Fig. 22.—Cord (Case 5), showing diffuse infiltration of anterior horns and vessels surrounded by cells.

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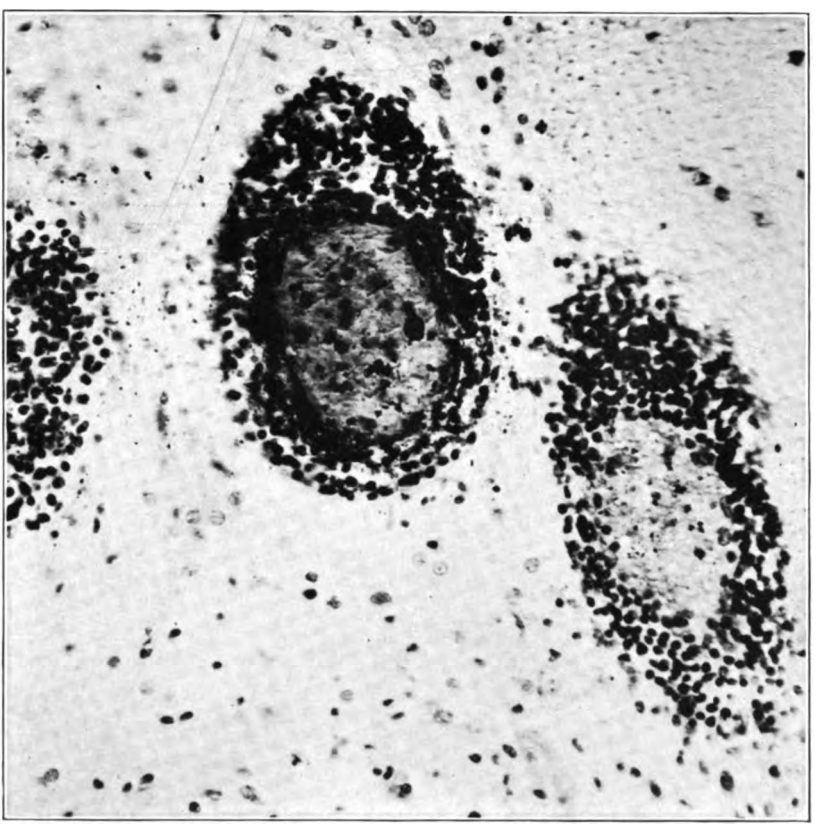

Fig. 23.—Perivascular collections of cells; enlarged photograph of vessels in Figure 22.

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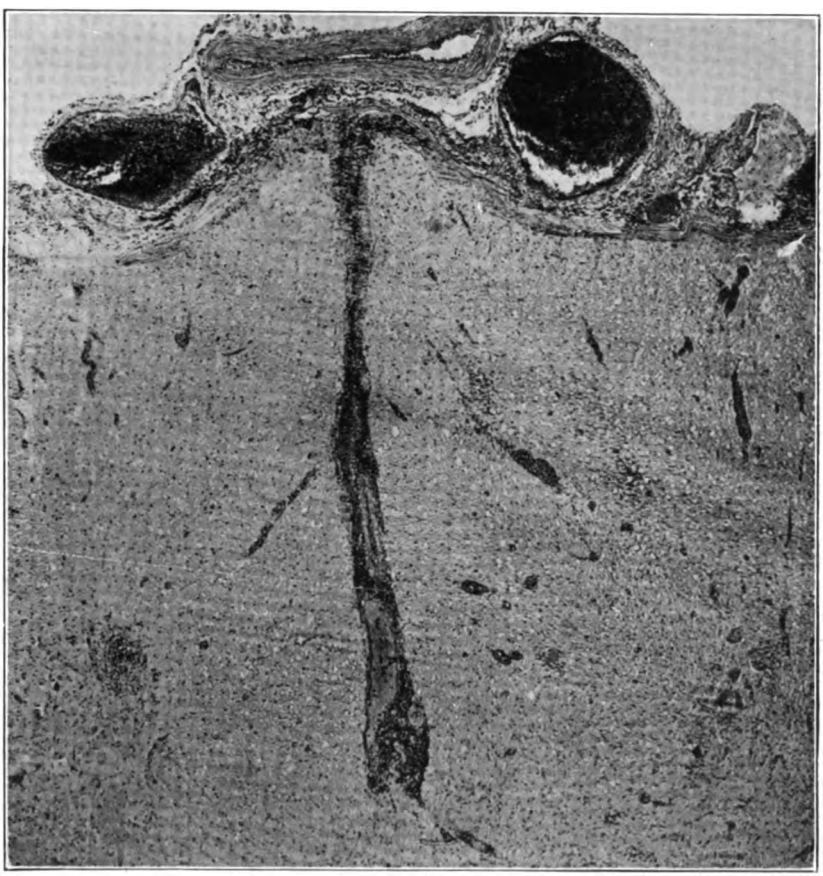

Fig. 24.—Anterior fissure and pia mater of cord (Case 6); congestion and infiltration.

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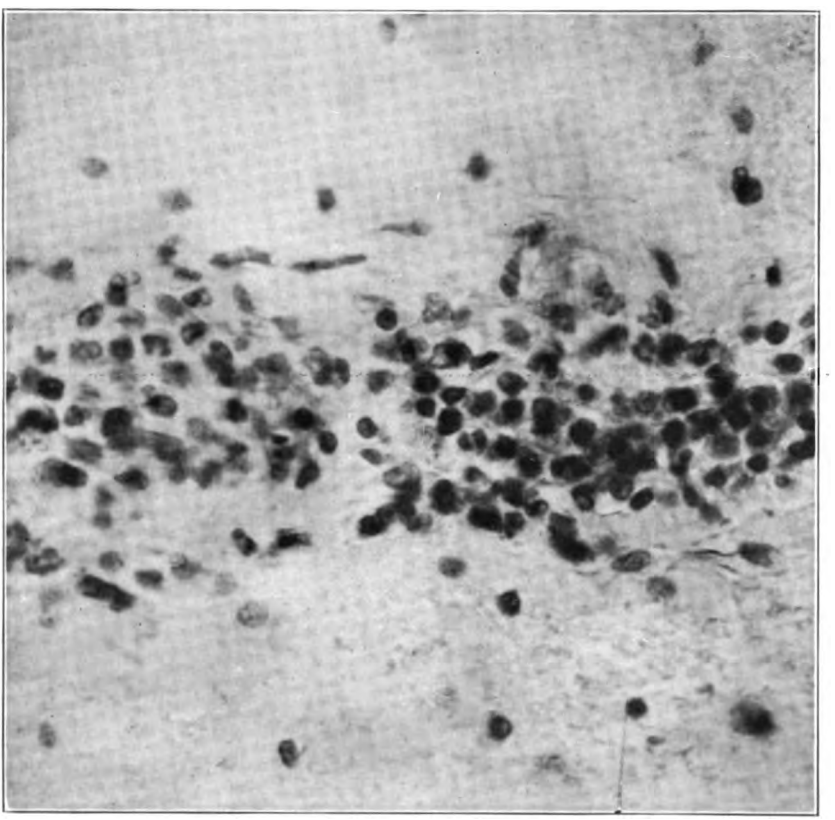

Fig. 25.—Cells in anterior fissure from Figure 24.

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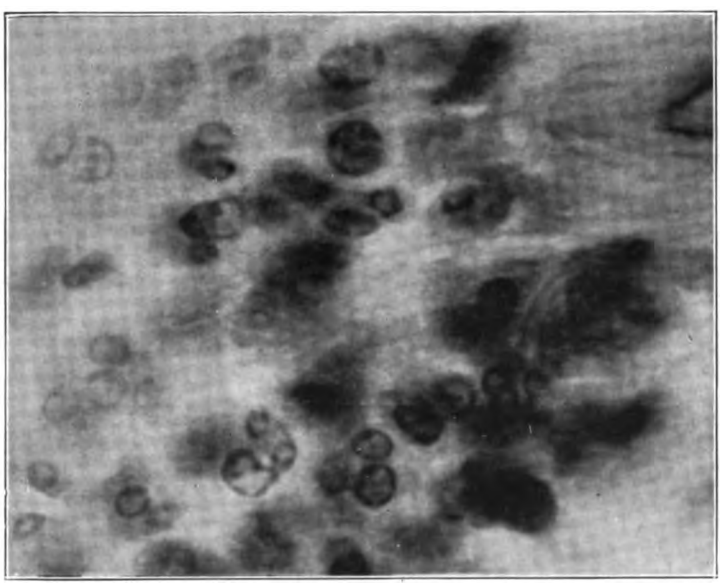

Fig. 26.—Polymorphonuclear leukocytes around vessel from Figure 24.

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Fig. 27.—Monkey inoculated with spinal cord from patient 5; paralysis following brain abscess.

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tained, they have not been the specific lesions of poliomyelitis. By isolation from mixed cultures this coccus has been obtained from eyes, noses and throats of tonsillitis, diphtheria and poliomyelitis patients and from the nose of a nurse caring for a child with poliomyelitis.

During the Pennsylvania epidemic, Drs. Fox and Rucker did some excellent bacteriologic work, which is given in detail in the Annual Report of the Commissioner of Health of Pennsylvania for 1907. Their description of the cultural and morphologic characteristics of Geirsvold's diplococcus is very complete and coincides exactly with our findings. Other references are purposely omitted, since the recent investigation at the Rockefeller Institute has surpassed all others in value and is a matter of current report.

In Minnesota, free examination of spinal fluid is part of the routine work of the Laboratory Division of the State Board of Health and specimens from twenty cases of poliomyelitis were examined during the epidemic of 1909. The results of this work are valuable as negative evidence, and some of the data relating to motor disturbances in inoculated animals and Geirsvold's diplococcus are as follows:

DATA OF SPINAL FLUID EXAMINATION

Spinal fluids from seven patients (five recovered; two died; no autopsies) showed bacteria obviously due to contamination. Several of the animals inoculated with these fluids died, but symptoms and lesions of poliomyelitis were absent. The other animals were well eight months after inoculation.

Spinal fluids from five patients (all died; no autopsies) were perfectly clear. The smears showed only a few lymphocytes. No organisms were found. A rabbit inoculated in the ear vein with 1 c.c. of one fluid remained well and gained 685 gm. in eight months. Was then killed and no lesions were found.

Geirsvold's diplococcus was found in purity in the spinal fluids from seven patients (two died; see Dr. Robertson's Cases 1 and 3; five recovered), and also in cultures from the blood, mouth, lung, spleen, kidney, middle ear and brain substance of patient 3.

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TESTS TO DETERMINE THE PATHOGENIC RELATION OF GEIRSVOLD'S DIPLOCOCCUS

Cultures from two of the patients recovering with paralysis, and from the two patients just noted who died, were grown for forty-eight hours on plain agar slants.

Eight rabbits of about the same size and weight were selected for inoculation.

Sterile physiologic salt solution was added to each slant and the cultures rubbed up to give uniform milky emulsions.

Each rabbit received 0.3 c.c. of a single emulsion. Each culture was injected into two rabbits, one rabbit being intravenously and the other subdurally infected.

One of the cultures killed both rabbits. The one inoculated in the ear vein died from acute pneumonia; the one inoculated subdurally died from meningitis, and, although the diplococcus was recovered in purity from his brain, no lesions of poliomyelitis were present in either case. The other six rabbits were not in the least affected by the inoculations.

It will suffice to state that twenty-eight rabbits, six guinea-pigs, two white mice and two rats were inoculated and, in spite of some symptoms of motor disturbances (amounting to complete paralysis in one animal), no specific lesions of poliomyelitis were discovered in sections of spinal cords. The spinal fluid of a rabbit dead of meningitis, caused by subdural injection of Geirsvold's diplococcus obtained from Case 3, was put into the brain of another rabbit. This rabbit developed a paralysis of the hind legs which progressed until he died on the forty-fourth day after inoculation from paralysis of respiration. He was greatly emaciated, his tendon reflexes were absent, and no response could be obtained by electric stimuli during the two days preceding death. The bacteriologic and pathologic findings were negative for the diplococcus and for lesions of poliomyelitis.

This case is cited to show how easily one might be led to misinterpret symptoms in an inoculated animal if the pathology of the case is not worked out carefully.

Our experimental work confirms the opinion previously expressed by other observers, that Geirsvold's diplococcus is not a causative factor in poliomyelitis.

In the meanwhile the production of experimental poliomyelitis in monkeys had been reported by Drs. Flexner and Lewis of the Rockefeller Institute. We secured from a kindly disposed owner of a nearby zoological garden two monkeys *(Macacus rhesus)* which had become acclimated to the cold Minnesota winter, and waited for another autopsy to give us

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some material for experimentation. The next post-mortem examination was Case 5 (See Dr. Robertson's notes), and it gave in cultures from the spinal fluid a mixture containing pneumococci. A terminal pneumonia readily accounted for the presence of this organism. Since a culture from the substance of the cord showed no growth after forty-eight hours incubation, the female monkey was chloroformed and 0.5 c.c. of a thick emulsion of spinal cord from patient 5 was injected into the brain substance on the right side. She had no trouble from the operation or the wound, which healed without suppuration in a few days. Not until the sixth day did she show any change in playfulness or appetite. For three days thereafter her appetite was impaired and her left foot and leg were not so warm as the other one. Then some paresis developed, chiefly of the flexor groups of the left arm and leg. She was quite sick for three days, although her temperature and leukocyte count did not fluctuate much more than before she was inoculated. The severity of her general illness lessened but the paralysis remained. Some loss of weight occurred and wasting of the flexor groups of muscles in the affected limbs became apparent. She was photographed on the twenty-second day after inoculation and then chloroformed, since she seemed to be suffering. A complete autopsy showed no macroscopic lesion in any organ except the brain. Here a large abscess took up about half the right hemisphere and from it the pneumococcus was obtained in purity. Microscopic examination of all organs proved the absence of lesions of poliomyelitis.

This sudden onset of a selective paralysis following a short period of general malaise might have been mistaken for acute anterior poliomyelitis had symptoms alone been considered. This emphasizes the previous assertion that symptoms in inoculated animals must be supported by pathological findings to have any value as experimental evidence.

The safety and utility of lumbar puncture as an aid in diagnosis and treatment should be borne in mind and the examination of spinal fluid should be a routine measure in public health laboratories.

We look forward confidently to the solution of this etiologic problem in the near future and wish the workers of the Rockefeller Institute victory over acute anterior poliomyelitis equal to their success in epidemic cerebrospinal meningitis.

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THE SURGICAL TREATMENT OF INFANTILE SPINAL PARALYSIS

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To one who thoroughly appreciates the pathology of anterior poliomyelitis the surgical treatment of the chronic stage of this affection yields satisfactory and gratifying results. A certain amount of irreparable damage has been done the spinal cord, in consequence of which the muscular power of the affected part has been correspondingly diminished. One may not hope to restore such a member to its normal condition; one may hope, however, to overcome deforming tendencies resulting from loss of muscular balance and to establish conditions such as will enable the part to functionate in the most efficient manner permitted by the existing degree of paralysis.

OBJECTS OF OPERATION

The objects of operation in this affection are two:

1. Improvement of form in the deformed member (by forcible correction, tenotomy, fasiotomy, myotomy, esteotomy, wedge-shaped excision of bone, and bone excavation).
2. Improvement of function in the non-deformed, or corrected; member.

A. Through restoration of power to the paralyzed muscle (by nerve anastomosis).

B Through restoration of muscle balance (by muscle and tendon grafting and transplantation).

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C. Through securing greater stability in the paralyzed member (by arthrodesis, astragalectomy, silk ligaments, tenodesis, fasciodesis, and removal of skin flaps).

I. IMPROVEMENT OF FORM IN THE DEFORMED MEMBER

It is a deplorable fact that, while deformity is generally conceded to be, as a rule, preventable by the early, proper and persistent use of passive motion and retention apparatus, the majority of patients consulting the orthopedic surgeon for this affection present malpositions of greater or less degree.

Deformity is most complex in its causation. One of the most essential factors is loss of muscle-balance resulting from unequal distribution of paralysis; it is well recognized clinically that a generally affected part may retain an approximately normal form, while paralysis of a single muscle or group of muscles, as, for example, the peroneals, may give rise to a severe malposition. To this loss of balance, which through unapposed muscular action permits of gradual lengthening of the affected and shortening of the unaffected muscles, are added the effects of gravity, of habitual posture and of functional use; finally structural adaptation takes place, fixing the part in the deformed position. When a malposition has been allowed to persist for some time, whether structural adaptation has taken place or not, a new factor, of great importance in the proper estimation of the degree of the paralysis, is added: this is the overstretching of the weakened muscular tissue. That damaged nerve cells may recover sufficiently to functionate, but in the meantime the affected muscles become incapacitated by overstretching and atrophy from disuse, is a fact[1] which, though well known to specialists, does not seem to be recognized by the mass of the profession and which certainly is very frequently disregarded by all in judging

1. Silver, David: Paralysis of the Shoulder, Am. Jour. Orthop. Surg., November, 1908.

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the results of operative measures. It is impossible by any tests at our command to judge accurately of the power of such overstretched muscles[2]; only by maintaining them in a position of maximum relaxation for a sufficient time to permit retraction to take place can we be definitely certain of their condition. Hence, any operation for the correction of deformity necessarily includes long-continued retention in an overcorrected position, and this, furthermore, must obviously precede measures for the improvement of function.

For any cases which have proved intractable to passive motion and mechanical measures a variety of surgical procedures—forcible correction, division of muscles, tendons, ligaments and fasciæ, and operations on the bones—are at our disposal, the choice of which will vary according to the mechanical conditions to be met, a combination of the various procedures being, as a rule, required.

Forcible Correction.—Forcible correction, performed manually or with the aid of various forms of apparatus, yields a uniform stretching of all contracted soft parts, permits the lengthening of deep-lying structures not readily accessible to the knife, and restores the muscle belly to its former length, instead of causing a further shortening and so a further decrease in excursion, as does simple division. With increase in the amount of force used, however, there is increasing danger of injury to soft parts and bones; moreover, under certain conditions one deformity may be increased while another is being corrected. One example of this will suffice: Given a case of talipes equinovarus, any amount of force applied to the sole can have no other than a beneficial effect on the contour of the foot, while in talipes equinovalgus, on the contrary, the force used to correct the equinus will necessarily tend to increase the valgus; hence, in this instance, operative lengthening of the tendo Achillis would be preferred.

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2. Tubby, A. H.: Recent Surgical Methods in the Treatment of Certain Forms of Paralysis, Brit. Med. Jour., March 1, 1906.

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Tenotomy.—While subcutaneous tenotomy after the original method of Stromeyer, when practiced in suitable cases, ordinarily yields entirely satisfactory results, yet in infantile spinal paralysis there are certain considerations which favor the choice of other methods. In stating these considerations the tendo Achillis only will be mentioned, as it is the one most frequently requiring division.

Simple tenotomy does not permit control of the extent to which the tendon is lengthened, yet this is a matter of considerable moment, for the strength of a muscle depends very largely on its tension, its strength being markedly decreased when the normal tension is lost, since a certain amount of force must be expended in restoring normal tension before joint movement can take place. Hence, in cases of corrected paralytic talipes equinus in which the tendo Achillis has been divided by the old method, it is not uncommon to find the power of plantar flexion much diminished, so that the patients are unable for months or even years to stand on the toes.[3] In an analysis of eighteen paralytic cases in which simple division of the tendo Achillis had been performed from three to eight years before, Hibbs[4] found serious modification of the function of the calf in eleven; he attributes this to the following factors: First, shortening as a result of the deformity; second, further shortening as a result of the tenotomy, and third, still further shortening from yielding of the bond of union between the divided ends. The control of the amount of lengthening is, therefore, advisable, and this may be secured through lengthening by the open method or by means of the subcutaneous method of Beyer.

Operations on the Bones.—Should bone changes have become so marked that forcible correction and division of soft parts are not sufficient to overcome deformity,

3. Lange, F.: Joachimsthal's Handbuch der Orthop. Chir., 1905-1907.

4. Hibbs, R. A.: Subcutaneous Division of the Tendo Achillis, etc., New York Med. Jour., July 10, 1902.

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this may still be accomplished through osteotomy, wedge-shaped excision, or bone excavation (Ogston's operation[5]).

While the procedures thus far discussed have for their primary object improvement of form, they necessarily contribute through this to improvement of function, and, when combined with the judicious use of apparatus and muscle development and training, will yield in many cases such satisfactory results that no further operative treatment will be indicated.

II. IMPROVEMENT OF FUNCTION IN THE NON-DEFORMED OR CORRECTED MEMBER

A. Through Restoration of Power to the Paralyzed Muscle (by Nerve Anastomosis)

The restoration of normal nerve impulses to the paralyzed muscle or muscles by means of nerve anastamosis would naturally be the operation of choice were it generally applicable to this affection. While the operation must still be regarded as in the experimental stage as regards anterior poliomyelitis, yet enough has been done to justify certain conclusions.

The operation has here evidently a more limited application than at first supposed. It is ten years since nerve anastomosis was first used in anterior poliomyelitis and five years since the subject was presented before this Association;[6] yet the successful cases available in literature are not numerous, in many, as is so often the case, the reports being made too early for the final results to be known. The reason for this limited field is to be found in the fact that the paralysis is rarely confined to a muscle group supplied by only one nerve-root or spinal segment, but is in the large majority of cases spread over several spinal segments, and so no nerve or nerve-root

5. Bartow, B.: Operative Remodelling of the Tarsus, etc., Am. Jour. Orthop. Surg., vii, No. 4.

6. Spiller, W. G., and Frazier, C. H.: Treatment of Acute Anterior Poliomyelitis by Nerve Transplantation, THE JOURNAL A. M. A., Jan. 21, 1905, p. 169.

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anastomosis can be beneficial. For a case to be suitable the lesion must affect an entire nerve-center, while other centers remain free.[7] Hence, cases in which the operation is indicated are undoubtedly fewer than previously considered.

While in the first burst of enthusiasm the procedure may seem to offer the possibility of ideal results, yet a study of the literature indicates that such is not the case. There is admittedly a certain danger of injury to the healthy nerve, and so, in the event of failure of the anastomosis, a danger that the condition following operation may be worse than that before; this may result from traumatism at the time of operation, failure of cut fibers to repair, or subsequent impairment from scar formation. Spitzy[8] admits that there is a loss in the power-giving nerve, but claims that it is not permanent. Stoffel,[9] in operating for restoration of the deltoid chose for the power-giving nerve the branch to the middle head of the triceps, since this could be sacrificed in the event of failure without great loss. Results further fall short of the ideal in that complete restoration of power, so far at least, has not been the usual result; this may be due to choosing a nerve that has itself been slightly affected, to imperfect regeneration in the power-receiving nerve, to injury from scar formation, or to failure of the new center to learn its function.

Early operation offers the best chance of success; the longer the duration and the fewer the signs of life in the affected muscle, the less the chance of recovery, for the muscle finally becomes changed into a fibrous, fatty mass from which restitution to the highly organized nerve-muscle organ is no longer possible.[8] Four to six months, in some cases nine months, is usually given as the most favorable time, yet operation as early as this is open to

7. Tubby, A. H.: Surgical Treatment of Infantile Paralysis, Brit. Med. Jour., Sept. 26, 1908 (Discussion).

8. Spitzy, H.: Fortschritte auf dem Gebiete der Chirurgie der peripheren Nerven, Wien. klin. Wchnschr., 1909, No. 46.

9. Stoffel, A.: Neue Gesichtspunkte auf dem Gebiete der Nerventransplantationen, Ztschr. f. orthop. Chir., 1910, xxv.

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the objection that the greatest possible degree of recovery has not yet been attained. Spitzy, perhaps the most ardent advocate of the procedure, admits that in a few cases recovery might have occurred without the anastomosis.

The newly developing function should encounter as little resistance as possible; hence overcorrection of deformity is a necessity, and this should be performed at a preliminary sitting for the reasons already stated. The combination of correction of deformity, lengthening and shortening of tendons, and even tendon grafting with nerve anastomosis at the same sitting, as has been reported, must be regarded as unscientific, since it is difficult under these circumstances to determine accurately to which procedure improvement is due; where nerve anastomosis is used tendon transplantation should be held in reserve as a secondary operation in the event of failure or of partial success of the anastomosis. As a further step in diminishing resistance to the returning function, alcoholization of the nerve-supply to the active opponents has been used by Allison and Schwab.

The upper extremity at present apparently offers the most promising field for nerve anastomosis. The hand, which on account of its highly differentiated function has not yielded such successful results after tendon transplantation as has the foot, seems for this same reason to offer more from a successful nerve anastomosis. At the shoulder, also, where there is no muscle capable of fully replacing the deltoid, conditions are admirable for anastomosis on account of the accessibility of the nerves, and a study of the reported cases shows that the operation in this region has given a relatively high degree of improvement.[9]

B. Through Restoration of Muscle Balance (by Muscle and Tendon Grafting or Transplantation)

The restoration of muscle balance by means of tendon and muscle grafting or transplantation has been so thoroughly tested that its efficacy in the treatment of the

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chronic stage of anterior poliomyelitis is to-day beyond question.

Tendon transplantation is no exception to the rule that every operative procedure has its limitations. In the beginning too much was expected of it. It brings no increase of power, except through subsequent muscular development; it merely permits the restoration of an important lost function at the expense of one or more less important ones, thus securing improved action in a part which is still a paralyzed one. If the paralysis be extensive the muscular tissue remaining will not be sufficient to ensure control after balance is restored; hence the method is obviously not applicable to cases of this character except when combined with other procedures.

In the early operations the use of slips of tendons often yielded nothing more than the benefit to be derived from their action as "additional ligaments," since independent action, without which there can be no restoration of function, was rarely obtained; moreover, relapse through gradual yielding of the bond of union or stretching of the weakened receiving tendon was common.[10] Not only have these causes of failure been overcome, but the usefulness of the method has been further extended through the transplantation of whole muscles or of parts of muscles readily divisable (triceps suræ, extensor digitorum, etc.), the use of silk extensions where the tendons are not of sufficient length, and insertion directly into the periosteum or bone at the most advantageous point.

The operative technic is comparatively simple and easily mastered by anyone acquainted with modern surgical methods, but the planning of the operation requires an intimate knowledge of the disease and an acquaintance with human mechanics which can be acquired only by special study. The lack of ability properly to determine the extent and the degree of the paralysis, defi-

10. Silver, David: Causes of Failure in Tendon Grafting, Pennsylvania Med. Jour., August, 1907 (References).

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nitely to estimate the amount of muscular tissue which must be transplanted in order to restore balance, and wisely to combine other operative procedures[11] with that of tendon transplantation, fully explain the failure of many surgeons to secure satisfactory results and make it certain that there will always be bad results.

The importance of an extended period of postoperative treatment, including especially protection of the transplanted tendon and careful muscle training,[12] has been so frequently emphasized that it needs no further mention.

As regards the adaptability of the operation to the various regions, the foot is preeminently the domain of tendon transplantation. Here the movements which are absolutely essential are few in number and relatively simple in character; of those required, plantar and dorsal flexion are of first importance, supination and pronation next, while control of the toes is not necessary in shoe-wearing people.[3] Hence, it will usually be possible, except where plantar flexion is lost, to secure a normally balanced foot; with lost plantar flexion, however, full restoration is impossible, since the combined strength of all the muscles on the back of the leg is only one-fourth that of the calf (gastrocnemius and soleus). Peroneal paralysis, one of the most common types, which gives rise to a talipes varus or equinovarus, is perhaps the most favorable condition in the foot for transplantation. In mild cases the extensor proprius hallucis may be transferred to the outer border of the foot, being attached to the cuboid; in marked cases, where the balance is more severely disturbed, the tibialis anticus may be used, if the posticus is normal; in this deformity weight-bearing is an aid in protection against relapse. Talipes valgus is not so favorable as varus on account of the greater power required for adduction and the

11. Whitman, R.: The Importance of Supplementing Tendon Transplantation in the Treatment of Paralytic Talipes by Other Procedures Designed to Assure Stability, Am. Jour. Orthop. Surg., August, 1903.

12. Lovett, R. W.: Treatment of Paralytic Deformities, Boston Med. and Surg. Jour., April 14, 1910 (Bibliography).

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deforming tendency of weight-bearing. In paralysis of the calf, transplantation alone will rarely do more than prevent a severe degree of calcaneus, but when combined with procedures which enable the muscles to act at a greater mechanical advantage excellent results are secured.[13]

At the knee the final results of substitution for the paralyzed quadriceps have been reported by Böcker,[14] as follows: Transplantation of the biceps and either the semitendinosus or semimembranosus, one flexor being left to prevent a genu recurvatum, gave the best results, almost normal extension with a good and steady gait being secured in most cases; the combination of the sartorius or the tensor vaginæ femoris with the semitendinosus or semimembranosus was less efficient, yielding only 100 to 130 degrees of active extension; the use of the sartorius or the tensor alone gave only moderate improvement, for, while it was possible for the patient to take a few steps on an even surface, apparatus was required for long distances.

In the region of the hip the results obtained by transplantation have so far been slight. Lange[15] has restored the power of abduction in complete paralysis of the gluteus medius and minimus by attaching the upper end of the vastus externus by means of silk strands, arranged in a fan-shaped manner, to the crest of the ilium; three out of four patients so treated were able to lift the leg when lying on the unaffected side, and the gait was greatly improved.

In the hand, where each muscle plays a definite part in the movements of precision here required, the restoration of any lost function can be purchased only at a high price; furthermore, independent action of the trans-

13. Whitman, R.: Further Observations on the Treatment of Paralytic Talipes Calcaneus by Astragalectomy, etc., Ann. Surg., February, 1908.

14. Böcker, W.: Endresultate der Sehnentransplantationen bei Quadricepslähmung, Arch. f. klin. Chir., 1909, xci, No. 1.

15. Lange, F.: Der plastische Ersatz der Glutaeus medius und minimus, Ztschr. f. orthop. Chir., 1906, xvii.

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planted muscle is essential and a longer period of muscle training is required than is the case in the restoration of the coarser movements of the lower extremity. This is sufficient explanation of the lessened efficiency of transplantation in this region. Moreover, on account of the less frequent affection of the upper extremity (one to eight as compared with the upper), opportunity for working out the problems here involved is much less.

The chief attempt in the upper extremity has been to replace the paralyzed deltoid. The difficulties to be met consist, first, in the fact that the different portions of the deltoid have different functions, and, second, in the lack of sufficient muscular tissue for substitution. A portion of the trapezius, the muscle usually chosen, will obviously replace the action of only one part of the deltoid; when this is supplemented by a portion of the pectoralis major a better result may be obtained, but full replacement would seem to be an impossibility. While the operation, therefore, has resulted in considerable improvement, even in some cases enabling the patient to elevate the arm above the shoulder, it falls far short of full function.

C. Through Securing Greater Stabilty in the Paralyzed Member (by Arthrodesis, Astragalectomy, Silk Ligaments, Tenodesis, Fasciodesis, Removal of Skin Flaps, etc.)

Arthrodesis.—When all the muscles controlling a joint are paralyzed, or in some instances in which the remaining power is not sufficient to ensure control, the operation of arthrodesis, in which the production of an artificial ankylosis is sought, is advisable in order to free the patient of all apparatus or permit the use of a less cumbersome form. Arthrodesis should never be considered until the use of those conservative measures known to be essential have been persisted in for a sufficient time to remove all doubt that the paralysis is permanent, nor should it ever be performed unless it is certain that the

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expected result will exceed the damage; that is, that the stiff joint will be a lesser evil than the flail joint.[16]

The adaptability of the various joints for the operation is in the order of ankle, knee, shoulder, hip, elbow, wrist; operation on the last two is seldom performed.

The operation is peculiarly suited to the ankle, on account of the character of its articulations and its weight-bearing function. It may be performed between the tibia and the astragalus alone, the usual procedure, or with this may be combined arthrodesis between the astragalus and the os calcis,[17] or at the mid-tarsus, the rigidity of the foot being thus varied according to the needs of the case. Bony ankylosis or strong fibrous union is necessary; the latter gives a more elastic foot but carries with it the danger of yielding under use. Operation on patients under 10 is not so likely to yield satisfactory ankylosis on account of the amount of cartilage present in the joints of those under this age.[18] When proper precautions are taken the number of failures is small, and results are both gratifying and permanent, the disadvantages being only those incident to the rigidity; that is, a lack of elasticity in the gait, some difficulty in walking on an uneven surface, and perhaps more early tire. In cases in which there is some muscle power left. but not enough to ensure control of the foot after transplantation alone, arthrodesis of one or the other joint may be combined with transplantation and will be of especial service where only fibrous ankylosis is secured.

At the knee pathologic ankylosis is so common that the advantages and the disadvantages of an arthrodesis of this joint will be apparent to all. The decision should be left to the patient; hence it is best not to perform the operation in children. The condition is so well controlled by apparatus which will permit bending of the

16. Vulpius, O.: Uber den Wert der Arthrodese, München. med. Wchnschr., Feb. 18, 1908.
17. Goldthwait, J. E.: An Operation for Stiffening the Ankle Joint in Infantile Paralysis. Am. Jour. Orthop. Surg., v, No. 3.
18. Jones, R.: Official Report on Arthrodesis, Internat. Cong. Med., Budapest, September, 1909.

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knee in sitting that this will usually be preferred, except in the very poor. Where both sides are affected, arthrodesis of one knee only is indicated.

While arthrodesis of the hip is not common, yet under certain conditions it is of distinct value, since muscle and tendon transplantation does not offer much in this region. Where both hips are paralyzed the production of ankylosis in one, which will usually be fibrous, will enable the patient to get on his feet. The method suggested by Albee for operation in osteo-arthritis should be a distinct addition to the technic of arthrodesis of the hip.

The shoulder, when the scapular muscles are unaffected, yields excellent results from arthrodesis. Vulpius,[19] who reports twelve cases, had only two failures, and obtained bony ankylosis, which is to be sought, in 60 per cent. The average active motion possible in his case was 75 degrees of forward elevation, 60 degrees of side elevation and 25 degrees of backward elevation; marked improvement in the atrophied biceps and triceps was observed.

Astragalectomy.—On account of its lack of muscular attachment the astragalus is a factor in instability at the ankle, and hence its removal may be indicated. Astragalectomy is one step in Whitman's operation[13] for paralytic talipes calceneus; when this is done the foot can be displaced backward, a procedure which gives improved stability and better leverage for the muscles which are transplanted into the heel.

Silk Ligaments, Tendodesis, Fasciodesis.—The chief objection to the older method of tendon grafting lies, as has been mentioned, in the fact that the improvement gained is often only that due to the action of the grafted muscle as an "additional ligament," independent action not being secured. Following out this idea, several methods have been suggested for directly utilizing the

19. Vulpius, O.: Die Arthrodese der Schultergelenks, Ztschr. f. orthop. Chir., 1908, xix.

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tendons of paralyzed muscles as ligaments, both as a supplementary operation and to obviate the necessity for an arthrodesis. The proximal ends of the tendons have been sutured to the bone (tenodesis of Codavilla-Reiner) or to the fascia (fasciodesis of Vulpius). The obvious objection to this is that the degenerated tendons will stretch and the method thus give only temporary improvement. The use of silk ligaments, a method at once simple and efficatious, yields a permanent fixation. These silk ligaments are of service in various joints but are in most common use at the ankle, where they are especially valuable in preventing toe-drop. In this condition, which may be taken as the type, the foot is simply suspended from the tibia by two double strands of silk; one double strand is quilted into the periosteum on the front of the tibia and then carried to the scaphoid, where it is sutured directly to the bone, while the other double strand passes from the same point to the cuboid.[20] Nature encapsulates the strands of silk and thus the ligaments are formed. Insertion directly into the bone must be at one end only to allow for adaptation in growth. This method of fixation may be used early in treatment, before other measures are indicated, for the purpose of removing all strain from weakened muscles and so giving them a better chance to recover; later in the course of treatment it may be employed when no muscle avaliable for transplantation is present and as a substitute for arthrodesis; it is also of frequent service as an aid to other procedures.

Skin Flaps.—A very useful operation for overcoming the tendency to deformity is the removal of skin flaps,[21] which recommends itself on account of its simplicity. If the anterior muscles of the leg are weak an oval piece of skin is removed from the top of the foot of such size that when the edges are approximated and accurately sutured

20. Scutter, R.: The Use of Silk Ligaments, etc., Boston Med. and Surg. Jour., June 4, 1908.

21. Jones, R.: Arthrodesis and Tendon Transplantation, Brit. Med. Jour., March 28, 1908.

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the foot will be held in the desired position of dorsal flexion; by slightly varying the location of the flap to one or the other side the foot may be inclined toward pronation or supination. This procedure has the advantage of not interfering in any way with any subsequent operation; as a means of retaining the foot constantly in the desired position and obviating the objections incident to brace treatment it is, therefore, applicable early in treatment and in very young children where other procedures are contraindicated. It may also be combined in other cases with tendon shortening and lengthening, and with transplantation or nerve anastomosis as an aid in holding the part in an overcorrected position. Similarly, at the elbow the removal of a skin flap is of service in holding the joint in flexion, being in this region preferred by its originator to arthrodesis.

CONCLUSION

With all these measures for the improvement of function, orthopedic surgery of the present has much to offer the sufferers from this pitiable condition of infantile spinal paralysis, and it is rare indeed to find an untreated case in which some benefit cannot be given. Fortunately for us, treatment is most efficacious in the region most frequently affected—the foot. Yet in all regions enough can be gained to more than repay us for all our labor. To secure the maximum improvement a wise combination of the various procedures will usually be required.

326 South Highland Avenue.

NOTE.—In addition to the authorities already cited, the following have been used:

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Hildebrandt, A.: Uber die Sehnentransplantation, Berl. klin. Wchnschr., 1908, No. 47.

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Lange, F.: Eine Verbesserung der künstlichen Sehnen, Ztschr. f. orthop. Chir., 1906, xvii.

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Murphy, J. B.: Neurological Surgery, Surg., Gynec. and Obst., April, 1907.
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Truslow, W.: Treatment of the Deformities of Infantile Paralysis, Long Island Med. Jour., September, 1908.

ABSTRACT OF DISCUSSION

ON PAPERS OF DRS. SILVER AND ROBERTSON AND CHESLEY

Dr. Archibald Church, Chicago: Every general practitioner must hail with delight the renewed activity of our surgical friends in the treatment of these very trying cases. My own experience is not very gratifying in the recommendations I have made for surgical operations in the correction of the deformities secondary to the inflammation of the cord of a poliomyelitic nature.

It has been the almost invariable result that after one or two operations either the patient, or his friends, or the surgeon becomes discouraged; and these operations have not been applied with sufficient frequency perhaps, to give the general operating surgeon much experience with, or much interest in them. The surgical fixation of a joint sometimes results very satisfactorily, especially in the knee or the ankle, yet I think astragalectomy unnecessary, because with very slight orthopedic apparatus the ankle-joint can usually be retained in a satisfactory position.

The recent recommendations of Dr. Schwab, carried out surgically by Dr. Allison, seem to promise something more satisfactory. They are not mutilating or destructive. By temporarily inhibiting the function by the injection of alcohol into the nerve supplying the muscles which have retained their function, and which tend to overact and so to produce the contracture deformities, one is enabled, according to the observations and experience of Allison and Schwab, to build up the power of the more or less atrophic, or completely paralyzed and atrophic muscles, and to secure better positions of the joints. This gives the opportunity for therapeutic massage and passive and active movements; so that the patient is led to put into play that fraction of power which has been preserved and which under ordinary circumstances is entirely overmastered by the contracture tendency in the stronger group of muscles. The technic is particularly simple. A nerve supplying the given muscles is exposed and alcohol in various strengths, 50 to 90 per cent., is injected directly into them.

With the transplantation of muscles my experience has been decidedly unfortunate. The functional gain has been of minimal value; but if we can in many of these cases obtain the enthusiastic assistance of a manipulator or massage operator, who will devote himself every day for a number of

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years to necessary manipulations, apparently severe and hopeless cases will frequently present a degree of recovery almost marvelous. I recall the case of a young girl who suffered quite extensive involvement of the upper extremities, trunk and lower extremities. Yet in the hands of an empirical rubber who calls himself a doctor, who has devoted himself to this case for five or six years, this girl has recovered practically after a time when I supposed recovery was out of the question. She now has a fairly plump pair of lower extremities, walks readily and sits up without orthopedic assistance. Some of her friends do not even know that she has been afflicted with a grave disorder. I attribute the results to the physician's attention and the local manipulations of the muscles by massage. Of course orthopedic apparatus had been applied and modified from time to time; various splints and braces have been devised; but the absolute devotion of this man to this case has produced results which I wish we might parallel in others, and thus gives me the courage to say that under such treatment, perhaps with the assistance of the alcohol injections, we may look for better results than we have attained hitherto, and without the necessity of serious surgical intervention.

Dr. L. H. Mettler, Chicago: I congratulate Drs. Robertson and Silver on their thoroughly scientific presentation of this subject. Dr. Robertson's paper indicates the beautifully logical way in which our knowledge has grown in regard to the nature of anterior poliomyelitis. We all remember how the disease used to be regarded as primarily an atrophic condition. In a word, the emphasis used to be laid on the wasting of the muscles and of the associated neurons. Then followed the suggestion that embolic and thrombotic obstructions in the terminal branches of the anterior spinal artery were probably the cause, with or without an accompanying inflammation, of the wasting of the anterior cornual cells. Now we have reached the point of recognizing the condition as a form of infective hemorrhagic myelitis, with the most destructive center of the inflammation located in the anterior horns. And finally we are beginning to suspect, on the strongest sort of grounds, that the infection is more or less specific in character.

It seems to me that this calls for a rewriting and reclassification of the disease in most of our text-books. Instead of referring to it as a systemic affection under the general impression that it is a unique and distinct trouble belonging to a particular functional tract of the cord, we should recognize it as merely a variety of myelitis. Abroad they are suggesting the advisability of dropping the "polio" from the name of the disease and calling it "focal myelitis." Whether we are prepared to make so radical a change in a name that has been so long in use may well be questioned. The use of the term "poliomyelitis" for general clinical purposes can do no

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very great harm if we are careful to keep always in mind that the disease is nothing but a form of infectious myelitis, with involvement, in some cases at certain stages of the process, of other parts of the card beside the anterior horns, namely, the white matter and the meninges. The atrophic changes, and their disastrous results, which so dominate the clinical manifestations of the disease later on, are more or less secondary or subordinate phenomena in the sum total of its pathology. These atrophic manifestations (and I am speaking especially of the lower motor neurons whose cell bodies nest in the anterior horns) are in all probability analogous to the pyramidal degenerations that occur in the cerebral palsies of childhood. The essential pathology of the infantile cerebral and spinal palsies, especially when due to infective hemorrhagic inflammation in the gray matter of the brain and cord, is in all likelihood the same and affords strong ground for the contention of Strümpell, more popular in Europe than in this country, that the differentiation of cerebral from spinal infantile palsy is a mere clinical differentiation dependent on the particular functional characteristics of the parts involved.

It seems to me very practical thus to lay the emphasis in infantile palsy on the infective hemorrhagic inflammation rather than on the later and subordinate degenerative processes. The former conditions suggests some better hope of managing the disease than does the latter. Infections and vascular troubles are always somewhat more amenable to treatment than are progressive degenerations. An early diagnosis, therefore, is more imperatively demanded under the newer conception of the pathology of anterior poliomyelitis; and if the theory be ultimately confirmed that the disease is due to some special germ or toxin, all the more imperative will be the demand for an early etiologic diagnosis; for in the early recognition of the nature and cause of the disease will rest the highest hopes of preventing its spread and further progress both in the individual and in the community. And finally, if we succeed in discoving a serum or other means of overcoming the specific toxin in this form of myelitis, infantile spinal palsy—and perhaps likewise infantile cerebral palsy in some post-natal cases at least—will not be regarded so hopelessly as it has heretofore been under the old atrophic conceptions of the disease handed down to us by Charcot, who was able to study it pathologically only in the late stages. Much will be accomplished in the future in the prevention and treatment of these cases if the bright indications in the newer conception of their pathology and etiology continue to develop.

Dr. P. Bassoe, Chicago: I am not surprised that Dr. Robertson finds no thrombosis in his cases, as that condition is exceptional. The question of meningeal involvement brings up the most interesting controversy that is now going on as to the mode in which the disease extends. Of course it is

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well recognized that in most of the fatal cases there is either an ascending or a descending infection. Most of the fatal cases present the picture of Landry's paralysis. According to Harbitz and Scheel there is a primary meningitis, very slight, with infection of the spinal fluid, and a secondary involvement of the cord along the vessels. Now, Wickmann of Sweden, who has examined about an equal number of patients, does not agree with this, although his findings are principally the same, but he interprets them in an entirely different manner. In all cases the extent of the inflammation is much greater in the cord than it is in the meninges, and it is greater in the central part of the cord, in the gray matter, and in the adjacent white matter, than at the periphery. So Wickmann says that inflammation occurs primarily in the gray matter of the cord. It is an infection with round-cell infiltration in the perivascular lymphatics, and it extends along the lymphatics up and down, so that the cause of the ascent or descent is not a successive involvement of different levels from the meninges, but an extension along the perivascular channels. Harbitz says that there are not enough lymphatic channels to account for that. The extension must be from the meninges. Now if in Dr. Robertson's cases there is a sufficient degree of meningeal infiltration to account for the extension, that would be of course in favor of Harbitz's theory; but if there are many cord segments in which the meningeal changes are minimal, it would be hard to account for the extension at all levels.

There seems to be no doubt that there are cases of this disease due to the same contagium in which we have only a meningitis; it is also true that there are cases of meningitis due to other organisms in which cord changes may be produced that are exactly like those in poliomyelitis. Attention has quite recently been called to that fact by Claude and Lejonne of Paris, and I have seen a man taken simultaneously with symptoms of meningitis and a flaccid paraplegia. That was three years ago, and I saw the man a week ago and he looked like a patient with an old case of poliomyelitis. If it had not been for the presence of the diplococcus found in the acute stage, I would say that it was an old case of poliomyelitis in which there had been considerable meningeal involvement.

Involvement of the white matter occurs in nearly all cases, and clinical evidences may be found if looked for. Only a week ago I saw a child of 14 who had typical flaccid paralysis of one leg, and spastic paralysis with increased reflexes of the other leg. I saw a man 30 years old taken sick suddenly with this disease six weeks ago, with a weakness of the left leg and the left arm. It came on with fever. After two weeks he could walk again, but he has retained an inability to raise the arm. I find that his left knee-jerk is increased, his arm reflexes are increased; he has an atrophic paralysis

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of the deltoid and the latissimus dorsi and pectoralis and supra and intraspinatus. It is a case of poliomyelitis involving that portion of the cord just above the level where the arm reflexes are represented. Neurath and others have called attention to the frequency with which we see a spastic condition of the leg in cases of poliomyelitis chiefly involving the arm. In many cases Neurath noticed the Babinski sign and ankle-clonus for a time while inflammation was going on. Later on the spastic signs in the leg disappeared and we had typical flaccid paralysis of the left arm, which shows that there was at the time an inflammation in the white matter, which subsided entirely.

Dr. A. L. Skoog, Kansas City, Mo.: I observed a number of cases last summer, while under appointment of the Kansas State Board of Health to investigate the Kansas epidemic of poliomyelitis acuta, both clinically and pathologically. One striking feature of the epidemic in Kansas was a grouping chiefly in bordering portions of two counties in the northwestern part of the state. There were also isolated cases throughout different regions of the state. One of the noticeable points of this epidemic was the high mortality rate, about 33 per cent. of the patients dying. All told there were about forty cases in these two counties. Another interesting feature was the number of cases in adults. Adult cases have been reported, especially by Wickmann and others. Wickmann reports a patient aged 67. In this epidemic I was in consultation on one case, that of a woman aged 45, who also had marked cerebral symptoms. She died on the sixth or seventh day of the illness and I was unable to obtain permission to make an autopsy, but I did examine the cerebrospinal fluid, which showed a lymphocytosis and a few polymorphonuclear cells, with a count of about 30 or 40 to the field of centrifuged sediment. The fluid was absolutely clear and contained no bacteria. No bacteria appeared on culture, on injection of guinea-pigs, rabbits, chickens and two rhesus monkeys. Another feature about this epidemic was the number of cases which puzzled the local physicians in making differential diagnoses from Landry's paralysis. In fact two physicians did diagnose Landry's paralysis; although from observation of this epidemic I believe Landry's paralysis may be considered as a form of poliomyelitis acuta.

In regard to lumbar puncture, it seems to me very important that we should obtain or look for some means of making an earlier diagnosis than we have in the past, and to me the examination of the cerebrospinal fluid has been attractive. I did a lumbar puncture in all the cases in which I could obtain consent, and that was about 7 cases out of 25. In all of them that I examined there was a mild increase in lymphocytes, ranging usually from 5 to 20 per centrifuge or

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sedimented specimen. The fluid was absolutely clear in all the cases examined, and I could find no bacteria.

I was especially interested in one point that Dr. Robertson brought out in regard to the ringing of the anterior horn cells. That showed very definitely a ringing of the periphery in several cells. I obtained post-mortem two specimens. One of those showed some ringing; and many of the cells showed marked ringing, very narrow however, around the nuclei of the cells. This ringing is a little different from the grouping of the large nissl granules so frequently described in the pathology of acute poliomyelitis, in that it seems to be a grouping of the finer granules.

Dr. Arthur S. Hamilton, Minneapolis: The Section is to be congratulated on having the opportunity of listening to these two papers.

I wish to refer especially to the last case Dr. Robertson reported, namely, the one in which, in a young individual, death was apparently due to bronchopneumonia and nephritis, and in which there had been some suggestion of poliomyelitis during life, and in which, in the cord, post-mortem, the changes of poliomyelitis were found.

As Dr. Bassoe has suggested, it has been shown that the cell changes found in poliomyelitis may be found also in other conditions in which there is an acute intoxication. In fact, as far as I know, it is quite impossible to tell the changes which occur in the large cells of the cord in one intoxication from those that occur in another. I have no doubt that in poliomyelitis most of the changes that occur in the cells are due to mechanical disturbance in the circulation, and the consequent upsetting of the nutritional balance of the cells; but I think we are safe in assuming that some of these changes are due to toxins, especially since the same changes can be found in other conditions where there is intoxication without mechanical disturbance in the circulation.

Six weeks ago there was admitted to the Minneapolis City Hospital a young man of 24, who passed through a very ordinary attack of lobar pneumonia, except that it was rather more severe than the average case. Aside from that there were no unusual features until, at the time when the man was about to sit up in a chair, it was discovered that he had no power in his legs. The physician had not observed it before, and the patient himself, though he had recognized the condition, had thought nothing about it, assuming that it was a natural consequence of his illness. When he came to get up he could not flex his thighs on the abdomen and had but slight power of flexion of the foot on the leg. There was no history of pain and, so far as I could make out, there was no disturbance of sensation. The patellar reflex and the Achilles jerk was absent. It did not look to me like a case of peripheral neuritis, although I find a few such cases

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have been reported as associated with pneumonia. There was very little subsequently of interest in the case and the man recovered quite rapidly. At the end of two weeks the reflexes had returned; he had obtained fair power in his legs; could stand without assistance, and in three weeks could walk out of the hospital, and has since fully recovered. It seems to me that this is a case of poliomyelitis, not in a technical sense, but in the sense that the lesion is much more likely in the anterior horn cells than in the peripheral nerves.

In reference to the change of name of poliomyelitis, it does seem as though the present term is rather misleading. At the same time I think we should hesitate to apply the term myelitis. It is true that the gross manifestation, as we see it, is a myelitis; but all the recent evidences show that we are dealing with a general intoxication, an infection of some sort. Dr. Robertson has brought that out in his statement of the post-mortem findings that there was evidence of acute intoxication in various other organs in addition to the changes which occurred in the cord. It seems to me, therefore, that we should wait for further light on the subject before we attempt another designation.

Dr. Nathaniel Allison, St. Louis: As orthopedic surgeons we are interested in these cases chiefly as the final results of a disease and we deal with final results, or hope to deal with them. The investigations recently made in the pathology of this disease makes it seem quite possible that in a few years there will be no more cases of poliomyelitis and no results of this disease to treat. This is a consummation much to be desired. Dr. Silver has gone over in a most systematic and complete way all the things that have been done surgically for the relief of poliomyelitis. He has taken the subject up in such a way as to leave very little to say about it, except in one particular, and that is the use of nerve anastomosis as a relief.

The selection of any operation for the relief of poliomyelitis must be based on a pretty wide experience and knowledge of the mechnical conditions to be brought about after the operation; many papers have appeared on this subject, and many reasons have been given for failure in tendon transplantation, for failure in arthrodesis, etc., and for failure following anastomosis. The work of Kilvington and other English experimenters shows that nerve anastomoses will regenerate and quite extensively. Dr. Schwab and myself have been working on these problems with the combined point of view of the neurologist and the orthopedic surgeon. We started to alcoholize the nerves in cases of poliomyelitis following the success we had in alcoholizing the nerves supplying muscle groups in spastic paraplegias and spastic quadriplegias. We have had some very successful cases in which there was overaction of the hamstrings, in which the alcoholization of the particular

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nerves supplying the overacting muscle group caused the overacting muscle group to cease to overact, and gave the weaker antagonistic group in spastic palsy a chance to pick up power.

Where there is sufficient power in a muscle or muscle group in the foot to produce a deformity, such for instance, as the deformity of club-foot, then there are active nervous elements in the leg. The question is to select these nerve elements and make what can be made out of them by nerve anastomosis, leaving the tendons and joints untouched. This should be considered always first as a procedure, because if regeneration is established the result will be better than that obtained by fixing a joint or by tendon or muscle transplantation.

We have done several operations of this nature, one for instance, in two cases of isolated quadriceps paralysis. The branch of the quadriceps was severed from the anterior crural trunk and put under the vessels into the obturator nerve. This has been followed by regeneration in both instances, and the opposing group to this return of power in this weakened quadriceps, is the hamstrings. The sciatic nerve was exposed down the back of the thigh and the branches to the hamstring muscles were injected with alcohol, thereby throwing out the opposing group for a more or less definite period of time.

The relative value of all the procedures properly performed has been clearly demonstrated in Dr. Silver's paper. Dr. Lovett has written a paper recently, in which he showed that failure was due to neglect on the surgeon's part of the after treatment of these cases. The patients are not cured when the operation is done. These are cases on which someone must put time, patience and much physical energy in order to secure the improvement that should be expected.

Dr. Frank R. Fry, St. Louis: We have all been hammering away at various modes of treatment for many years, and as Dr. Church has intimated, with very little encouragement. In more recent times it seems to me the important function of the neurologist in these cases is to impress on the patient and those who have charge of them the fact that they have a chronic situation on hand for the treatment of which they must stir up as much enthusiasm among themselves as they possibly can in the midst of adverse circumstances. For instance, massage, I believe, is uniformly accepted by neurologists as the only rational routine method. We should tell our patients that massage must be practiced not for a few months but for several years. Many patients cannot afford to have the services of a professional rubber; we must educate somebody in connection with the case to carry on this work effectively.

Some years ago Dr. Hammond, of New York, taking a lot of old cases of poliomyelitis in which muscles showed no reactions whatever to electrical current, was able, continuing

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the current with persistency, to develop reactions in these muscles. He published this fact, but he never published the sequel of it, which I obtained in personal conversation with him, namely, that afterward these muscles lapsed to their former condition. That is the kind of discouragement to which we have become used. It is on account of that fact that neurologists to-day are more interested in what the orthopedists are doing, than in anything they feel capable of accomplishing themselves. I personally feel no comfort in trying to take care of a poliomyelitis patient without being in contact with a reliable orthopedist all the time.

Dr. C. R. Woodsen, St. Joseph, Mo.: I believe that the lack of success of orthopedic measures is due largely to the fact that too many men undertake to do this work, who are not familiar with the nervous system, especially its anatomy and the function of various nerves. There are few men who do this work well, and there should be a specializing along that line.

There is one thing that every physician who sees a case of poliomyelitis can do and should do, and that is, to make an early diagnosis. A lack of effort to diagnose is responsible in many instances for a failure to diagnose, but the important thing to do is to prevent deformities. It can be diagnosed before paralysis appears. It may not be diagnosed of course, until afterward, as has been stated, but the important thing is to retain a proper temperature and prevent deformity and institute early treatment of massage and electrotherapeutics. It is easy to prevent deformities if the case is taken early. In some cases treatment does not restore function, but the prevention of a deformity makes the cure much more easy.

Dr. C. H. Hughes, St. Louis: Having had a rather extensive experience in the direction of poliomyelitis anterior, as you may probably guess from the color of my hair, I wish to make an addition of something that has not been contributed to this subject.

Two instances are now in my mind of recoveries from poliomyelitis anterior. One child was brought to me in the arms of the nurse, sent to me by a very able physician down there in southern Illinois, where malaria is said by the inhabitants to be so thick that you could cut it with a knife. I noted that there were no cerebral symptoms connected with the case; this absence is to be expected in a typical case of poliomyelitis. There was no delirium; there had been no coma at any time; it was not a case of cerebral apoplexy; both lower limbs were paralyzed, and it occurred to me, being familiar with the practice of medical men in this section of the country and below, that I would add to my treatment a vigorous course of quinin. Quinin and protoiodid of mercury

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were sufficient to bring about a complete restoration in that case of poliomyelitis anterior within six weeks without deformity. I considered the cure complete in ten weeks and permitted the child to go home. That girl is now a grown woman, free from any evidence of any sort of the after-consequences, contracture deformities, etc., that so often confront us, to our dismay, in the management of poliomyelitis anterior. Unfortunately I have not had the good fortune in the majority of my cases to see such satisfactory results effected.

In the other case the child was placed by the nurse on the steps on a hot summer day and fell asleep; and when the nurse came to it again the child had no use of its lower limbs, but had not lost consciousness, had no mental impairment, and no evidences of anything else except the involvement of the anterior horns of the spinal cord. That child recovered, and that is all the recoveries out of a good many cases that I might record.

Notwithstanding the microbic invasion in poliomyelitis acuta, we generally believe that its attack is somewhat similar to that of pneumonia. Now the common impression is that the pneumonia develops the chill. My impression is that the chill permits the microbic invasion, and then we have the work done by the pneumococcus in pneumonia and the infective agent in this essential paralysis of children thus gets in its damaging work.

Dr. H. E. Robertson, Minneapolis: In regard to the involvement of the pia mater I am unable to agree with the authorities who urge that such involvement is primary. I have always found the involvement of the cord more marked in any particular region than that of the corresponding pia. The place of infection is the perivascular lymph-channels, extending to the interstitial portions of the gray matter of the cord, and second, to the lymph-channels in the pia mater, and even to the gray and white matter of the posterior columns.

Dr. David Silver, Pittsburg: My paper was intended as a review of the literature of the present-day surgical treatment of this affection. Regarding Dr. Hammond's results, cited by Dr. Fry, one would naturally expect relapse, because, while muscular tissue was present, it was not sufficient to meet the demands of functional use. In such cases some procedure is obviously required to decrease the amount of work demanded of the muscles, as for example, arthrodesis.

An unusual amount of patience is required in dealing with these cases. I mentioned several procedures which seem to me of distinct value and I wish again to emphasize them. My first point is that in a drop-foot, for example, it is obviously of much greater value, early in the treatment, to fix the foot by taking out a skin flap or by making an artificial ligament,

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which does not interfere with any subsequent operation, than to fix it with a brace, because the brace not only interferes with the function of the part but its daily removal is a disadvantage; the part must be retained constantly in the position of maximum relaxation, if a return of power in the weakened muscles is to be secured. The second point is that in every case deformity must be overcome a long time before any operation for the restoration of function is attempted. Usually when the patient consults the surgeon, some operation like nerve anastomosis or tendon transplantation is at once proposed, instead of preliminary treatment being instituted to determine accurately the degree and the extent of the paralysis and to put the part in the most favorable condition for such operation. If such preliminary treatment is carefully carried out, I am sure our results will be very much more favorable.

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INTRADURAL TUMOR OF THE SPINAL CORD WITH OPERATION

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REPORT OF CASE AND ANALYSIS BY DR. MAYER

The diagnosis of tumors of the spinal cord is no longer difficult, nor is their operability in a majority of cases a debatable question. Indeed the large number of patients successfully operated on has fully justified the optimism of Victor Horsley as expressed by him three decades ago. Since then in every country many have contributed to our knowledge of tumors of the spinal cord—the recent books of Malaise, Bruns, Oppenheim and Schlesinger covering well the entire subject—so that we shall try to avoid being too discursive.

CASE REPORT

Clinical History.—July 12, 1909. Family and previous history are not noteworthy. The patient, M. T., had not had syphilis, gonorrhea, or any infectious disease, but probably did overexert himself and subject himself to cold and privation. A cloth manufacturer in Russia, he was reduced to poverty, and for some years after coming to America peddled, carrying a fifty-pound grip. For the last four years he had been a storekeeper in a small way.

Present Illness.—In 1904 he felt a slight pain between his shoulder-blades. This was continuous for about one month and was especially noticeable when he threw his head back or turned his neck; when recumbent he could move head and neck freely without any pain manifesting itself. This pain grew worse, was intermittent, and would shoot down the inner side of his arms, and at one time disappeared for over a year. In October, 1908, on the advice of his physician, he went to Mount

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Clemens to try to get relief there from this pain. At this time he could not stoop to tie his shoe laces on account of the pain in his neck. His spine was at no time stiff, however, nor was there any lateral deviation or torticollis. When he would sit for any length of time, a numb feeling would steal up his legs which he would not notice when he was standing or walking. Later, a similar sensation developed around his right elbow. Pain between his shoulder blades not only persisted, but now would not stop when he was walking or when recumbent or with any position of his head. While at Mount Clemens, he took the baths and massage and believes his pains disappeared for a few weeks. However, he returned home very weak, and a little later, (November, 1908,) noticed a weakness in his right leg which could not be lifted as easily as before. This grew worse from day to day. About a month later, his right arm began to feel heavy and his hand-grasp was weaker than formerly. This weakness increased to paralysis. About January, 1909, his left leg and arm became involved similarly. He came to see me on January 11, was told of the seriousness of his condition and the probabilities of an operation, and advised to go immediately to the hospital. He chose to be guided by other opinions, however, and presented himself again only on July 12, six months later.

Examination.—At this date the patient was absolutely helpless, having been bedridden from the beginning of March. He complained very much of cramps in his calf muscles, accompanied by involuntary flexion of his legs, with sharp pains extending up his arms and back, to his neck. He had absolutely no power of voluntary motion in his lower limbs. When his lower limbs contracted involuntarily, his fingers drew up also in the ulnar side. He had no erection and no ejaculation for six months, nor had he any longer control of either bladder or bowel. When he wished to cough or take a deep breath he felt a constriction or sense of heaviness across his chest. The patient slept soundly except when awakened by the spasms. There was no disturbance of hearing, smell, taste or of speech. There was absolute paralysis of lower limbs, with pronounced clonus, and incontinence of urine and feces.

Right Hand and Forearm: Weakened power of supination; no power for extension of hand; inability to flex fingers completely. He could flex second phalanges about thirty degrees. There was inability to abduct thumb; slight abduction and adduction of fingers on radial side; absent on ulnar side. Interossei and intrinsic hand muscles were slightly atrophied. Hand grasp was zero.

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Left Hand and Forearm: Extension and flexion weakened but neither so much as on right side; abduction of fingers weakened; hand grasp zero.

Reflexes: Mendel-Bechterew phenomenon present; knee-jerks both much increased; Babinski present in both legs; Oppenheim present in both legs; Gordon present in both legs; cremasteric reflex, faint; abdominal reflexes, absent; triceps and ulnar reflexes, diminished; scapular reflex, absent. On extreme lateral movements of eyeballs there were slight nystagmoid movements. Right pupil slightly larger than left. Normal pupillary reactions. Equal palpebral fissures. Fundi normal. Normal neck and shoulder movements. No enlargements of vertebræ. Entire spinal column freely movable. Percussion, which was taken with posterior surface of the flexed index-finger elicited slight tenderness over sixth cervical vertebræ. Auscultation with percussion (tonal percussion) over vertebræ negative.

Sensation: Patient complained of a cold pain in right leg. In areas of lost sensibility, patient felt at some places a delayed sense of touch or pain without being able to orient it; that is, to tell where he had felt it. This was most noticeable in the lumbar flanks and inguinal region. Lost tactile sensation extended one segment higher than lost pain sensation, as high as the second rib in front and the first dorsal vertebræ behind. Temperature sense was disturbed about equally in areas shown by chart, but especially on the right side of the body. Inner side of arms anesthetic, analgesic and thermanesthetic; bathyanesthesia and pallanesthesia of both lower limbs.

Wassermann negative; von Pirquet negative; blood examination negative; radiograph negative.

Diagnosis.—Tumor involving seventh and eighth cervical and first thoracic spinal nerves, the sensory changes pointing especially to the eighth cervical and first thoracic, and the muscle changes to the sixth and seventh roots as well. Operation was not arranged for until September on account of taking our vacation.

This history on analysis read so clearly that a diagnosis of spinal cord tumor was easily made, its location, structure and extent only requiring a further study. It is needless to emphasize the importance of establishing these points in discussing the question of a spinal tumor and its operability, which carry with them a consideration of the segments of cord or of spinal roots involved.

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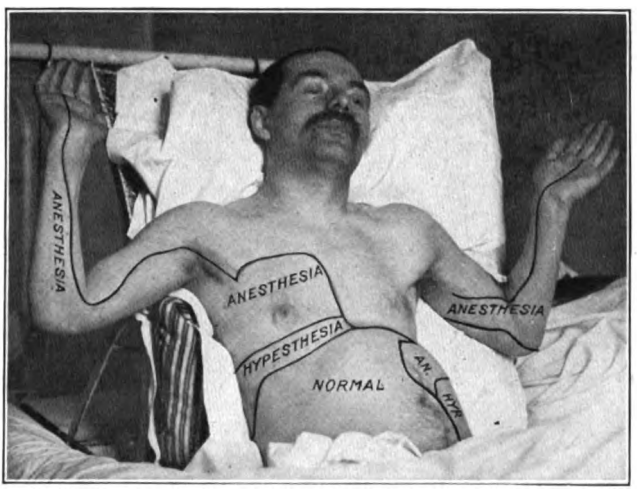

Fig. 1.—Patient with intradural tumor of the spinal cord, front view, showing areas of anesthesia, hypesthesia, and normal sensation; from photograph made four months after operation.

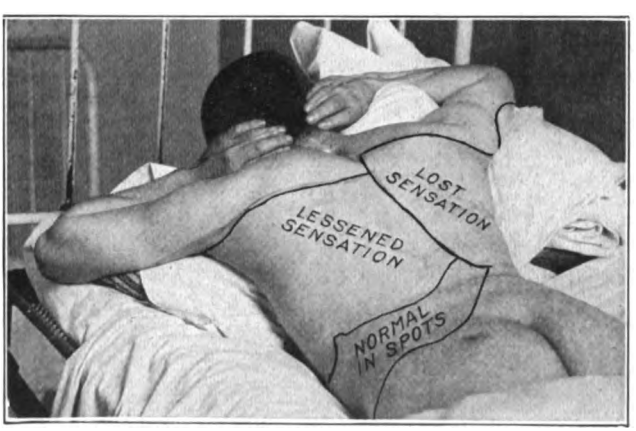

Fig. 2.—Same patient, back view, showing areas of lessened sensation, lost sensation, and normal sensation in spots; from photograph made four months after operation.

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I am omitting all special points connected with tumors of the vertebræ themselves, or of intramedullary tumors, except as they enter into the question of the diagnosis of intravertebral tumors, extradural or intradural, which are primarily the operable types, and whose clinical histories are practically similar. It is to the intradural variety that this case belongs.

SYMPTOMS AND DIAGNOSIS OF INTRADURAL TUMORS

Symptoms of intradural tumors, following somewhat the arrangement of Bruns may be grouped as follows:

1. Spinal Roots:
    a. Irritative symptoms.
    b. Compression symptoms.
    c. Atrophic symptoms.
2. Spinal Cord:
    a. Symptoms of local pressure.
    b. Symptoms of tract disease:
        1. Unilateral.
        2. Bilateral.
    c. Symptoms due to growth of tumor into cord.
3. Vertebral:
    a. Erosion.
    b. Trophic.

In other words, depending on the origin, size, and manner of growth, intradural tumors will slowly but progressively evoke symptoms of unilateral, later bilateral, pain and hyperesthesia, radicular in type, spasms and then paralysis, perhaps of a Brown-Séquard type at first (unilateral compression of cord), but generally becoming rapidly a paraplegia (total compression) with atrophy.

With Mr. T. no objective early history was obtainable, but even his meager description brings out a very important fact; that is, the fairly constant pain between his shoulder-blades was made worse on movement. Though there are exceptions, whenever an algia of such a location is intensified by movement of the head or by stooping, coughing, etc., a strong suspicion of spinal root irritation should be instantly created. Indeed, if at that time, it had been found on examination that the algia

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extended segmentally, and not according to a peripheral innervation, with the absence of any lateral deviation of the spine or stiffness, a probable diagnosis of spinal cord tumor at that time, even before the onset of any paresis atrophy or segmentary anesthesia, would, I believe, have been justifiable. The necessity of examining for objective sensory changes twice a month or oftener, is also indicated, in order to detect the oncoming of this significant diagnostic point. The occurrence of edema in the innervation zone of the painful spinal root, to which Schlesinger and, lately, Pearce Bailey call attention, is a valuable help when present. With doubtful pains, corsets and braces or extension are advocated, though Oppenheim calls attention to their making the symptoms rapidly worse. The question of an eccentric projection of pain (Shultze) should never be forgotten, and all possible diseases of the thoracic and abdominal organs excluded. An extension upward of a spinal growth would of course, involve new segments and roots and evoke symptoms higher up the cord. An extension downward need not cause any new symptoms; never, if there has been a complete conductive paralysis. A bilateral pain is more often found in vertebral tumors than in tumors of the roots and spinal cord, and if in addition shortening of the spinal column is noticed, carcinoma of the vertebræ is most probable. Of course, the incomplete intercostal neuralgias which are not of Head's type, found in hysteria and the presence of trophic muscular disturbances accompanying simple chronic neuritis, must always be excluded.

The fact that three nerve roots must be affected before distinct sensory and motor paralysis occurs emphasizes in my opinion the importance of a careful investigation of these prodromic symptoms of spinal root irritation, their intensity, constancy and stubborness to medical treatment, their aggravation on movement and explosive respiratory acts, the segmental character of the pain and tenderness or hypesthesia present and possibly

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localized tenderness or increased pain on pressure over vertebræ. A change in the vertebral percussion note manifestly could occur only late in the growth of the tumor (except of the vertebral tumors), and if it did occur early, should awaken suspicion of a serous meningitis more than of tumor. The change, however, in the percussion note is so slight that it can never be of much value as a diagnostic factor. Palpation and percussion should always be done slightly to each side of the spinous processes and in palpation I have found that pressure upward helps to get a more reliable result. Percussion on one side and listening with the stethoscope on the other side of the spinal column is a good method.

A striking point in our patient's history is the length of his period of pain. Though the course of spinal cord tumors is a slow one, and of the intradural type the slowest (an average of twenty-six months according to the most reliable statistics), with Mr. T. we have a history of four to five years of radicular pains, indicating possibly an irritative state before the appearance of, or without an increase in the size of, the tumor. The question, moreover, of a possible primary radicular disease preceding the growth of the tumor, must be considered. Such a protracted course is, however, not exceptional as the histories given by Horsley, Bruns and Oppenheim show, though in our patient it persisted longer than in the patients reported by them before compression symptoms developed. I could hardly reconcile this slow course with the pathologic finding of an endothelioma.

We have learned from experience that motor symptoms without sensory alterations, even though they increase progressively, cannot be accepted as conclusive evidence of the presence of a tumor of the spinal cord, and that whenever there is reasonable certainty of its presence, the place for operation depends on the localization of the upper pole of the growth. Charting on the skin

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the areas which show sensory alterations is without question necessary in every case of spinal cord disease, and especially in suspected tumor cases. Further realizing that every muscle and skin area is innervated by from two to four segments, we must locate the seat of cord injury at least one segment higher than the paralysis of motion and sensation indicates. I have, for instance, at present in my service at the South Side Hospital a probable case of dorsal-cord tumor in which the motor paralysis is of the second to the fourth lumbar segments and where the upper pole of the hypesthesia is at the level of the tenth dorsal vertebra. Operation will be justified, however, a little higher, as it is reasonably sure that the seat of trouble is higher. The pains and spasms of this patient are particularly prominent and call attention to the importance of such symptoms in localizing the upper pole of the growth. Indeed, where this so-called anesthesia dolorosa, or the so-called paraplegia dolorosa of Cruvelhier, is present, its value for localization exceeds every other symptom. Though therefore in this latter patient we would from her paralytic symptoms view her paralysis as lumbar, the hyperesthesia and spasms point to a higher innervation and must be considered in the operation to be performed on her.* Another interesting symptom of this patient is the intermittent presence of Babinski's sign, present one day, absent the next.

Unless the surgeon is also a neurologist, he should always have the seat of injury or of disease thoroughly located, if possible, before operation. I was, for instance, recently asked to examine a patient because he did not recover motion and sensation after a laminectomy; in him I found segmental symptoms several segments higher than the operative site, though I could not assert, not having examined him previously to the operation, that there had not been an ascending myelitis since

* Since this was written, an operation on this patient was performed and no tumor but a localized meningitis was found.

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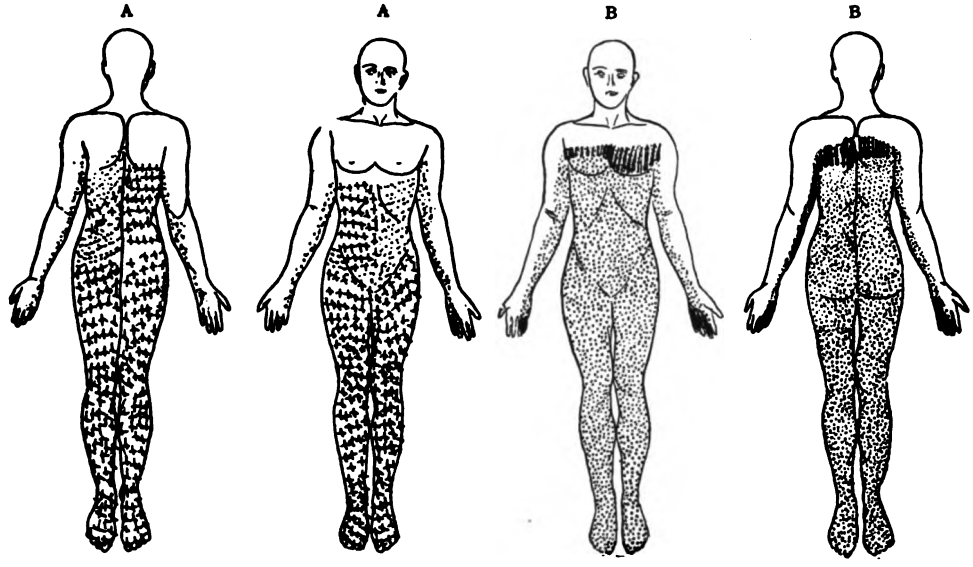

Fig. 3.—Diagrams of sensation areas in same patient before operation. In A A, area covered with crosses indicates loss of temperature sense to both heat and cold; stippled area indicates deficient temperature sense, either delayed or not always correctly interpreted. In B B, stippled portion indicates the region in which both pain and tactile senses are lost; portion shaded with lines indicates additional area with tactile anesthesia; solid shaded portion in hands represents lost muscle sensation.

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the operation. How much better, however, had it been if this patient had been thoroughly examined before operation!

To return to our patient who is the subject of this paper: The sensory charts revealed an involvement of the last cervical and upper dorsal segments. We assumed the upper pole of the growth to be a little higher, because of the total anesthesia and paralysis, and because of the local tenderness ignored the dorsal cord entirely, and the operation, skilfully performed by Dr. Gaub, revealed a tumor at the sixth cervical segment. I wish to also add that Drs. McKennan, Diller, Henniger and Silver saw this patient and confirmed our diagnosis.

One cannot too strongly remember that to have paralysis, two or three spinal roots or segments must be injured, and that with an area of intense pain the spinal root above, or perhaps two spinal roots, are probably affected. Individual variations of course occur, so that whenever we have hyperesthesia lying segmentarily over an anesthetic zone, it clinches localization. In segmental localization the upper pole of the lesion is always higher than the symptoms indicate and often higher than the area laid bare by the surgeon. That the innervation zones are given by some authorities a segment higher or lower than generally accepted can be practically ignored, because of the rarity of such an occurrence, coupled with the fact that we must, on account of the trisegmental innervation spoken of before, and of the topographic relations of cord, roots and vertebræ, allow for an error of at least one segment. I am not forgetting that dissociated anesthesia as a temporary symptom has been occasionally reported even in extradural tumor, but it is an extremely rare occurrence.

Given a positive segmental diagnosis in any progressive disease where the pains are severe and there is no hope for any other kind of benefit, operation, in spinal cases, is indicated (except where gliosis symptoms are

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present), even where the diagnosis is not clear, and subacute myelitis, localized serous meningitis or pachymeningitis have not been absolutely excluded.

Postoperative History.—The damage to the cord in our case was too extensive to warrant absolute recovery, though Mr. T. is better and his life has been saved. Loss of sensation gradually decreased to a hypesthesia on the right side and his spastic phenomena measurably decreased in intensity, but not entirely. Both motion and sensation gradually improved in his left arm and hand, but not in his right, and he can now turn in bed, partially sit up and move his toes, all of which was impossible for him to do last summer. The last sensory chart made of him shows that his thermic sense has practically become normal on his right side; that the upper level of the hypesthesia is not as high as was the anesthesia (right side), but practically the same on the left side. His right hand remains totally paralyzed. Notes made by my assistant, Dr. MacIntire, this spring read:

"Right Arm: Partial abduction of wrist. No abduction. Palmaris longus causes flexion of wrist. Slight contraction of flexor sublimis digitorum seen in movements of index finger only. Spade-like hand. Thenar and hypothenar eminences atrophied. Pronation and supination normal. No lateral movements of fingers or of thumb. Left Arm and Hand: All muscles normal."

The pictures taken by Dr. MacIntire show the present sensory involvement. A last report from our patient informs us that he now sits up ten hours daily and sleeps soundly all night, his obstipation being what most troubles him at present.

REPORT OF OPERATION BY DR. GAUB

The patient was placed in the prone position, with the head slightly lower than the shoulders, which eliminated the normal curvature of the posterior surface of the neck.

A straight incision was made over the spinous processes of the vertebræ, beginning at the fourth and ending at the first dorsal spinous process. The soft tissues were removed from the spinous processes and lamina of the fifth, sixth and seventh cervical vertebræ.

The arches of the sixth and seventh cervical vertebræ were removed by means of Macewen's bone-cutting forceps. Nothing abnormal was found. The dura presented a normal appearance and pulsated.

At the suggestion of Dr. Mayer the arch of the fifth cervical vertebra was removed. The dura was opened in the

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median line, revealing the cord, whose contour showed no gross alteration.

The exposed cord, at its lower level, felt resistant on palpation. By means of an aneurism needle the cord was gently drawn to the left, which exposed a mass the size of a Malaga grape, bluish white in color, situated on the right anterolateral surface of the cord, apparently traversed on its posterior surface by the posterior root, on account of the dislocation of the cord, and on blunt dissection it seemed to surround the anterior root of the sixth cervical nerve.

During the process of removal the tumor ruptured, but with care it was removed, possibly with the exception of a minute portion of the growth, which may have remained in the sixth intervertebral foramina.

The tumor on microscopic examination, made by Dr. Proescher, pathologist to the Allegheny General Hospital, proved to be an endothelioma. The dura was closed with interrupted catgut sutures, the soft structures, as far as possible, coaptated by means of a continuous catgut suture, and the intermuscular space drained with rubber tissue.

The patient was placed in bed on his side. There was moderate shock. Within a few hours, the temperature rose to 102.6 F. and continued a degree lower, with slight remissions, for the next seven days. There was moderate leakage of cerebrospinal fluid. After the removal of the drain, the patient suffered a chill and a rise in temperature to 103.2 F.

The temperature dropped to normal within thirty-six hours. It continued at 99 F. for a week, when there occurred a second chill, moderate in severity, and a rise in temperature to 101 F. This quickly subsided and the temperature remained around 99 F., until four days later there was another chill and the temperature arose to 102.6 F., which again quickly receded.

Two more chills, mild in character, and an increase in temperature occurred at intervals of four days, following which the temperature remained normal.

During this time there was moderate leakage of cerebrospinal fluid through the drainage tract. The patient was maintained in the semiprone position for three weeks and then gradually allowed to lie on his back. The surgical convalescence from this time on was uninterrupted.

Several points in the operative technic for the removal of tumors of the cord are engaging the attention of surgeons at the present time.

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The conservation of cerebrospinal fluid is of great importance. Attention given to the position of the patient, both on the operating table and when returned to his bed, the minimizing of drainage and the accurate suturing of soft tissue would tend to serve this purpose. It is probably better to close the dura with a continuous suture of catgut.

Careful hemostasis, accurate suturing, and coaptation of the soft structures, tend to close the hiatus left following the removal of the arches, and minimizes the necessity for drainage, but the surgeon may feel it safer to insert a rubber tissue to care for any dead space between the muscle layers.

After the dura is opened, avoidance of frequent mopping with gauze will save cerebrospinal fluid.

A tumor hidden by the cord is best recognized by palpation. In the present case pulsation was present, no gross alteration of the cord presented, and the presence of a tumor was determined by gentle palpation.

The question of a two-stage operation, as advised by Elsberg,[1] for intramedullary growths, appears to me to be a wise procedure. For the intradural type of tumor, its removal, as a primary procedure, is preferable, provided the patient's resistance warrants the completion of the operation.

A study of the literature presents the surgery of spinal cord tumors in a favorable light and it may well be that patients will submit to operation when a tumor of the cord is strongly suspected by competent diagnosticians, and not postpone the operation until irreparable damage to the cord has been done.

ABSTRACT OF DISCUSSION

Dr. Theodore Diller, Pittsburg: Through the kindness of Dr. Mayer I was permitted to examine this patient before the operation and also to witness the operation. I can bear witness that the examination was thorough and complete, and

1. Elsberg, C. A.: The Extrusion of Intraspinal Tumors, The Journal A. M. A., April 16, 1910, p. 1308.

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that the tumor was found as was expected and in accordance with the markings of the diagram which is going around the room. I must join with him, too, in his regret that the improvement in this patient is not greater, and that the case did not come to operation at an earlier time. I believe that Dr. Mayer is right in attributing the lack of improvement to the fact that the operation was so long delayed. Secondary degenerations must have occurred.

Dr. Julius Grinker, Chicago: About a year ago it was my privilege to report a case similar to this one, and it was published in The Journal and in the transactions of this Section. Dr. Mayer's experience was almost identical with my own. The diagnosis had not been made until it was too late to produce a perfect cure. The patient recovered and has regained sensation fully, but not much motion. Recently I have again seen the patient and found her quite comfortable, but she cannot walk. She has a typical spastic paraplegia with contractures. In that case the localizing diagnosis was correct and the operation was a success, but the true nature of the disease had not been recognized sufficiently early. She had had neuralgic pains for a long time, and was treated for intercostal neuralgia before I saw the case.

Recently I had a similar case in which I made a diagnosis of tumor of the spinal cord and localized it correctly. What was supposed to be a tumor was found opposite the eighth dorsal vertebra. That case, however, proved to be a cyst, a so-called meningitis serosa circumscripta, a number of cases of which have been recently reported by Munro. I do not see how it is possible to correctly diagnose the pathology of a tumor before operation. In my presentation of the case before the Chicago Surgical Society, I mentioned the various kinds of neoplasms and also the possibility of finding a cyst. The operation was performed by Dr. S. C. Plummer, of Chicago, who discovered a circumscribed cyst and no tumor. I did not feel that it was a mistake in diagnosis. Munro has called our attention recently to the little known fact that in diagnosing spinal tumor we must always make the reservation that it might be a cyst rather than a tumor. In the event of it being a cyst, it should be emptied of its contents; and this would be equivalent to the removal of the tumor, because we cannot dissect out the very fine membrane without injuring the meninges. My patient made a good recovery from the operation. Diagnosis was based on the fact that there was an abrupt beginning of the line of hypesthesia and hypalgesia; there were no continuous pains; only occasionally some neuralgic pains on one side. The opposite side showed a monoplegia or spastic paralysis of the Brown-Séquard variety. Sensory disturbances on one side, motor disturbances on the other, and an abrupt beginning of hypesthesia and hypalgesia, enabled me to diagnose neoplasm rather than transverse myelitis. There were sphincter disturbances which have im-

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proved since operation; the patient walks better; sensation is slowly returning, and it appears as though a perfect recovery will ensue.

Dr. Charles L. Dana, New York: The surgery of the spinal cord has certainly made great advances in the last few years, and the success in the removal of tumors of the spinal cord has been very much greater than that of removing tumors of the brain.

In New York we have had in the last five or six years a great many operations of this kind, and it has been shown that a very large proportion of these tumors are operable. If you look over the classified list of the tumors of the cord as given in the text-books and monographs, you would not suppose there were many tumors that could practically be removed. As a matter of fact in the cases that I have seen operated on they are nearly all fibrosarcomata or endotheliomata, as the pathologists now choose to call them. And they are pretty nearly always either extramedullary or growing from the pia and not infiltrating the spinal cord to that extent that they cannot usually be dissected out.

The diagnosis of spinal tumors has been made of course, more exact; but in addition to that, exactness of diagnosis has not been so necessary as formerly, for the reason that the operation for exploration has been rendered comparatively safe. Dr. Taylor has advised an operation for unilateral laminectomy by which we can very quickly and easily cut down, expose the spinal cord, and if there is no tumor, or if the conditions do not warrant operation, the parts are restored, and the patient is pretty well in a comparatively short time; and for that reason sometimes we do not have to postpone the decision of operation for so long a time as we otherwise would. I always insisted on the niceties of diagnosis. You cannot by any possible means always, it seems to me, say that it is a tumor. I think in about 90 per cent. of cases we can be quite sure, but in the other 10 per cent. we are likely to run into a cyst, or sometimes a certain rather curious thrombotic softening of the cord, which is found occasionally instead of cysts.

I have found in one or two cases that the *x*-ray has been of help in diagnosticating the location and seat of the tumor, especially when there is any involvement of the bone in connection with the disease.

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HEMIPLEGIA FOLLOWING ACUTE INFECTIONS

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AND
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MINNEAPOLIS

The cerebral palsies of childhood have received considerable attention in certain rather elaborate treatises; such as those of McNutt, Lovett, Osler, Sachs and Spiller in this country, and of several writers of abroad, but aside from these not much has appeared on the subject. Thus, even in the most elaborate systems of medicine, paralysis of central origin in diphtheria is given the briefest consideration, or else is not mentioned at all. These facts are thought to justify the publication of the following reports of cases.

CASE 1.—*History.*—The patient, a schoolgirl, aged 9, was seen at the University Free Dispensary Feb. 8, 1908. Her father was living at 38 and well except that he was nervous; her mother was living and well at 36. She had had two miscarriages, both at about three months. Two brothers and one sister were living and well. A paternal aunt was paralyzed at 24, following labor. Another paternal aunt was extremely nervous. Otherwise the family history is negative. The patient's general health had been good. She was the second child. There was no trouble at her birth. She had croup in early childhood; had never had convulsions, but when a child had a habit of holding her breath when crying. She walked and talked at the usual age, and did as well in school as others. She was never seriously ill up to her attack of diphtheria, and was never seriously injured.

Present Illness.—In September, 1906, she had a severe attack of diphtheria with membrane in her throat and nose. She was given 12,000 units of antitoxin and seemed to progress

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ordinarily well until the middle of her attack when she developed darting pains in the right arm and leg. They were spasmodic, and each one lasted only two or three minutes. For four days the pain grew steadily worse and finally was very severe. It then began to decline and at the end of the week was practically gone. When present, the pain was worse in the arm than in the leg. There was no tenderness to touch between the attacks, so far as the mother recalled. The mother thought that even during the pain the arm and leg were rigid, and certainly they were so afterward. There was diplopia and some trouble in articulation for a short time following this attack. During the intervening two and one-half years up to the present time there had not been much change. The right arm was spastic with frequent more or less spasmodic contractions. The leg had been less spastic, but was still noticeably stiff, and the patient had slight trouble in walking, and considerable trouble in running. Attention always made the condition worse.

Physical Examination.—The patient was a moderately well-developed, well-nourished girl. The temperature was normal, the pulse 86. The forehead was somewhat low and narrow. The eyes and ears were normal, except for a slight tubercle on the right ear. The vault of the hard palate was high and narrow and very irregular. The teeth were set very irregularly. The arteries were soft. The apex-heat of the heart was in the mid-clavicular line. Otherwise the heart examination was negative. The right leg seemed slightly smaller than the left. The right arm was very firm and muscular and, evidently, better developed than the left. The measurements were as follows: The right forearm, a little below the elbow, 17 cm.; the left forearm, a little below the elbow, 16.75 cm.; the right calf 22.75 cm., and the left calf 23.5 cm. The patient was subject to frequent attacks in the right hand and arm of an athetoid character. The attack started in the hand with hyper-extension of the hand and flexion of the wrist. The hand was rotated until the palm was turned completely outward, when there was a flexion in turn of the second, third and fourth fingers. The arm was then rotated in the opposite direction until the palm pointed inward. The entire right arm and leg became spastic during these maneuvers. There was no pain associated with them. The patient had increased knee-jerks, especially on the right side, but there was no ankle-clonus. There was a possible faint right Babinski. She seemed to feel cheerful and happy and was always active, although at times she said she was unusually tired. She had a little frontal headache occasionally.

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Course of Disease.—Directly after coming to the dispensary the patient was placed on thyroid extract, 5 grains twice a day, and within a few days her arm and leg had made a very striking improvement—so much so that her parents were convinced of her complete recovery at an early date. The cause of this improvement was not easy to determine, but it was probably not due to the use of the thyroid extract, especially since, after three weeks, there was no further improvement, and within a short time there was a complete relapse to her former condition. She continued coming to the dispensary at more or less long intervals up to about the end of 1908. At that time there was no permanent improvement, but there had been several short periods of temporary benefit. On Dec. 19, 1908, when waked in the morning, she appeared unusually drowsy. When eating her breakfast she said that her face on the right side felt numb and later that it felt as if needles were sticking it. After a little time this feeling went to her right hand also. Finally, all the right side felt the same way. This condition of affairs lasted until 10:30 a. m., when the feeling began to go slowly away, but the patient was very sleepy for some time afterward. After the feeling had partially gone, the mother noticed that the hand and arm had lost their spasticity and were scarcely up to normal so far as tonus was concerned. There was also some lack of power in the hand and arm. On the afternoon of the first day of the numbness the patient was brought to our office at 3:30. She was then a little dull and heavy mentally, and there was still some feeling of numbness in the right side. The reflexes were about normal on both sides. The right arm hung rather limply by her side. There was absolutely no trace of spasticity or athetosis and all movements of the right upper extremity were performed with less power than those of the opposite side. There was some headache on the first day, and in the evening this became very severe. The next day she was stupid and weak, but when seen on the following day she seemed as well as ever, except that the condition of spasticity on the right side had returned. Shortly after this time she disappeared from observation and we have not been able to learn anything of her since.

CASE 2.—*History.*—The patient is a single woman, aged 22. The father is living and well at 51; at times drinks to excess. The mother is living at 47 and has heart trouble and tuberculosis. One sister died of diphtheria. One sister and four brothers are all well. There is no other nervous or mental trouble in the family, but a number of the mother's family have died of tuberculosis. The patient was always delicate but up to 12 years of age was never seriously ill. She had

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measles in childhood, but never had rheumatism. She walked and talked at the usual age. There was no trouble at her birth. She was uncommonly bright up to the present attack.

Present Illness.—At 12 years of age she had "black diphtheria." She lived twenty miles in the country and had no physician until after her paralysis appeared, and no antitoxin at any time. Three or four weeks after her attack of diphtheria she suddenly developed a right hemiplegia, including the face, arm and leg. There was a complete loss of motor power, including speech. Apparently there was no sensory involvement. The condition lasted one day, when she appeared to entirely recover and remained well for one week at the end of which time there was a second attack with very much the same symptoms and lasting two days. At the end of two weeks she had still a third attack, following which she was in bed for six weeks. As this history is given by the mother from her recollection, it is not certain that all the facts are accurately stated, including especially the complete recovery between the attacks. The mother says that the patient was not unconscious at any time, but that her mind after the third attack was "just like a baby's", by which she probably referred to the fact that the patient found it necessary to relearn speech. At the end of twelve days she could move her foot a little, and at the end of seventeen days spoke the word "Henry." The mother thinks that at this time she could understand spoken, but not written words. As time passed she gradually acquired control of the right side, first in the face, then in the leg, and later in the hand and arm. Her face apparently recovered completely. There has always been a slight stiffness in the right leg, and the right hand and arm are distinctly spastic. She was a long time in learning to speak, but finally reached the point at which she had very little trouble, though even at present she cannot always recall words as she wishes to. She went to school after recovering from her paralysis and learned readily. She is always cheerful and happy in disposition.

Physical Examination.—The patient is a well-developed and well-nourished young woman. There are no evidences of degeneracy. The pupils are equal and react normally to light and distance. The ocular movements are normal and there is no nystagmus. Vision is good, but the patient says that occasionally when looking suddenly to one side she has double vision. Smell and taste are normal. There is no involvement of the fifth or eighth nerves, but there is a slight defect in the right lower seventh. Her tongue is normal in appearance and is protruded straight. There is no trouble in swallowing. The heart and lung examination is negative, except for a faint

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blowing mitral murmur. The arteries are soft. The urine examination is negative. The deep reflexes of the left arm are normal; those of the right are much increased. The right patellar and Achilles reactions are very active, the left patellar and Achilles are normal. There is no ankle-clonus, but there is a faint Babinski reaction on the right side. The patient walks and runs well, but complains of slight stiffness in the right toes in movement, and there is a marked degree of spasticity in the entire right upper extremity. Grip in the right hand measures 17 and in the left hand 27. Muscular power in the right biceps and triceps is fair, but not so good as in the left. The middle finger and the thumb of the right hand are the most difficult to move. The first and little fingers are the easiest to move. The right forearm at its largest point measures 21.25 cm., and the left at a corresponding point measures 22.75 cm. Nothing unusual is noted in the patient's mental condition, except that, contrary to her mother's statement, she does not appear to be a particularly bright girl. Her speech is normal, except that she occasionally has some difficulty in finding the proper word.

CASE 3.—*History.*—The patient is a boy, aged 4. The father is living at 38; well and temperate; mother living at 37, well. There are no brothers or sisters. There is no tuberculosis, cancer or nervous or mental trouble in the family. There was no special trouble at the patient's birth and instruments were not used. The patient was breast-fed. His general health has been good, except that he has had croup every winter. At two and one-half years he had measles, and one year ago had diphtheria, but was not very ill with either. His appetite has been good, and his bowels regular.

Present Trouble.—On Dec. 28, 1909, the patient became ill with lobar pneumonia involving one lung. He was very ill and his temperature was above 103 for nine days. Three days after his temperature went down he developed a right hemiplegia. At noon he was noticed to have some weakness in the right hand. This slowly increased, and by the next day his hand and arm were completely paralyzed. Later in the same day the leg and foot of the same side also became paralyzed and there was motor aphasia. The face was not affected then or later. There was no sensory disturbance noticed and no disturbance of the sensorium. Thirteen days after the paralysis appeared he began to move the hand and arm, and speech power began to return, and in a few days he could put the hand on top of his head. He now uses the hand as well as ever, and his enunciation is good. A few days after power of movement appeared in the arm he began to use the leg, but improvement was slow, and even now use of this limb

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is decidedly impaired. There were no eye symptoms at any time. Three days after the paralysis appeared he developed a left middle-ear abscess which broke spontaneously and is now quite healed.

Physical Examination.—The patient is a well-developed and well-nourished boy. His head is well formed and there are no scars or evidences of degeneracy. The temperature is normal, the pulse 94, strong and regular. The examination of the heart, arteries and lungs shows nothing abnormal. The tongue is clean and is protruded straight to the front. The abdomen is protuberant. The pupils are equal and react normally for light and distance. The ocular movements are normal, and there is no nystagmus. Sight and hearing are good. Muscular power in the right arm seems normal. The patient limps slightly with the right leg, but examination shows no muscular weakness of any consequence. The circumference of the right thigh and calf is slightly less than at the same point on the other side. The reflexes in the left arm are normal, in the right moderately increased. The left patellar and Achilles reflexes are greatly increased. There is a slight ankle-clonus and Babinski on the right side. The abdominal and cremasteric reflexes are active on both sides. Sensation is normal everywhere, so far as can be determined. The right fingers are a very little stiff. The right leg and foot are moderately stiff. There is a very slight ataxia in the right hand. Speech and mental condition are normal.

CASE 4.—*History.*—The patient, a girl of 11, is one of ten children. The family history is good in all respects. The patient has always been considered well and strong, though not large for her age. There is no mention of any of the ordinary children's diseases. Since 5 years of age, she has attended school and has done well there.

Present Illness.—The trouble is dated from Jan. 4, 1910, though the patient had not been quite natural for the two preceeding weeks and had seemed to want to be alone and undisturbed. On Jan. 4, 1910, in the afternoon, she suddenly fell over on her side or back. She was unconscious at the time, but remained so for a few minutes only. The same afternoon she was able to walk, but the right hand and arm were almost paralyzed. She attempted to speak, but was unable to do so. She seemed to understand what was said to her, and appeared perfectly conscious. There was no muscular twitching and no pain. At about 8 p. m. she began to talk and her speech was entirely normal, until the next morning at 8 a. m. when there was again the loss of speech power without the other manifestations of trouble. For three days and nights this alternating loss and return of speech power continued, since when she has

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been unable to utter a word, except "papa" and "mamma." From the date of the onset of her trouble to February 1 she was about the house most of the time. Her mind seemed clear and she comprehended all that was said to her. She attempted to play with the other children, but could not speak. During this time, however, she had occasional "sinking spells" when she would fall, always toward the right side. She always attempted to protect herself in the fall. She was not unconscious, and there were no symptoms resembling convulsions. She slept well at night; had no rash or other sign of illness. During all this time, except the first three days, her mental condition was good. Her appetite was poor. There was no involvment of the right leg at any time, and up to January 29 the improvement in the hand and arm was so marked that she could sweep a room with very little difficulty. The parents noticed no disturbance in the eyes or face. On February 2 at 1 p. m. clonic spasms developed in the right side, involving, at first, the face, shoulder, arm and hand, and toward evening the right lower extremity. Each attack lasted two or three minutes, and was followed by a short period of drowsiness, and then by another attack. The parents think she was unconscious during these attacks, but not during the interval. During the afternoon and night of February 2 and all day of February 3 she was never long free from a convulsion. Toward evening the attacks became a little milder and a little less frequent, but on February 4 she had convulsions about every ten minutes. She complained of no pain, and gave no signs of distress. She could swallow, but apparently had no desire for food. She had little, if any, fever (parents' statement).

She was admitted to the University Hospital February 4 at 8 p. m. Her temperature was then 100.6, pulse 120 and respiration 24. When first seen by one of us the following morning she was lying asleep. There was nothing unusual in her attitude or appearance, except that the right corner of the mouth drooped a little, and there was some fullness of the right upper eyelid. Directly after being awakened a convulsion began, starting in the right face. The eyelids then began to twitch, the right more than the left. The right eyelids during the convulsion were closed and the left about half open. The convulsion then extended to the right arm and then to the right leg. The whole attack lasted about one and one-half minutes. During the next thirty minutes six more attacks occurred, four being general and two limited to the face only. Subsequently they occurred in about the same order of frequency up to the time of her operation. Both facial attacks lasted fifty seconds.

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In all the attacks, both eyes were drawn strongly to the right. The pupils were dilated and did not react to light in the midst of the most severe attacks, but did so in the lighter spasms, and toward the end of the more severe convulsions.

All the attacks began in the face save one, which commenced in the right arm. Usually the order was face, arm, leg, and cessation of movement was in the reverse order. She was probably conscious throughout the light attacks and at the end of the more severe ones, but in the midst of a severe convulsion, sharp pricking of a pin produced no response. During the severe attacks the right rectus abdominis muscle was very hard, the left only slightly contracted. The convulsion never spread to the left side, except that the left eyelids twitched decidedly and in the most severe attacks the left arm and leg contracted slightly.

The reflexes varied decidedly. After a severe convulsion no reaction could be obtained in the right arm or leg. This included the plantar reflex, which could not be demonstrated at any time in the right foot during the examination. In the interval between attacks the facial and abdominal reflexes appeared about normal. The deep arm and leg reflexes were somewhat increased, and, though varying considerably from period to period, there was no striking inequality in the two sides, except directly after the convulsion. There was no evident voluntary movement at the time of the examination in the right arm or leg, but on the day preceding the patient was observed on one occasion to move the right arm slightly.

In speaking or swallowing there was no movement in the right face. On request the patient closed the eyes and wrinkled the brows normally. A subsequent careful examination showed very slight response to light in the pupils in the midst of a fairly severe attack. A possible slight Babinski was obtained on the right side. As far as could be told there was no sensory disturbance. She swallowed well.

At times the patient spoke the words "yes" and "no" quite distinctly. She occasionally called the word "nurse" and said "good" for food that pleased her. She never used a complete sentence. She probably comprehended what was said to her and sometimes attended to requests and sometimes did not. She displayed a childish interest in a tie-pin; at times asked for a bed-pan. She screamed repeatedly as if in severe pain.

Dr. Baker, the resident physician, had observed double deviation of the eyes, and had repeatedly seen elevation of one eyeball and depression of the other, with frequent variation and, toward the end of a convulsion, a peculiar oscillatory movement most marked in the right eye.

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Urinary examination previous to the operation showed nothing of consequence, except specific gravity of 1036 and a few cylindroids. Two days after the operation a trace of albumin, but no cylindroids or casts, was found. A few days later even the albumin had disappeared. Previous to the operation the leukocyte count was six thousand. The temperature was variable, but frequently up to 103.

Operation.—On February 7 an operation was done by Dr. J. E. Moore under ether anesthesia. A large bone flap was removed from over the left motor area. When exposed the cortex showed some congestion and slight bulging of the brain substance. Beyond this, in a careful examination, nothing was found. The flap was replaced and the skin sutured.

Postoperative History.—On February 10 there was no movement of the right hand or arm, but a very slight movement in the right foot on deep pricking. Sensation seemed greatly lessened in the right leg and foot, but was probably not wholly absent. The mental condition was much better than it was preceding the operation. At 7 p. m. following the operation, the patient had a convulsion lasting two minutes. At 8:15 there was another, very slight, lasting only a few seconds. In the next hour there were two slight convulsions, but at one time she was conscious and answered when spoken to. Up to 7 the next morning, however, there were forty-five convulsions but all much milder and of much shorter duration than previously. The convulsions continued to 8 p. m. February 9, gradually growing less severe and less frequent. Early on the morning of the 10th the patient again had several slight convulsions, but none afterward. When she was sufficiently recovered from the operation it was found that she had a partial sensory aphasia and also visual amnesia.

The wound did nicely until February 16, when the patient pulled her dressing off and infected it, which greatly delayed its healing. Otherwise her surgical convalescence was uneventful, and the return of motor and sensory power in the right side was fairly rapid. No culture was made at the time of the operation, but when suppuration appeared in the wound the *Staphylococcus pyogenes aureus* was obtained from it.

On March 4 the patient moved her right leg very well; movements of her hand and fingers were also much improved, and she was able to pick up an orange with this hand. On March 16 she was up and walked about. March 23 she walked quite well, except that the right leg was rather spastic. The right arm was greatly improved, but the grip in the hand was feeble. All movements with this arm were performed slowly and were both spastic and ataxic. There was no special

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atrophy of hand, arm or leg. Sensation for touch, pressure and pain was apparently normal in the tongue, face and extremities. In speaking and laughing the right face behaved about the same as the left. At this time she was also beginning to speak and write a few words and could understand the name of most objects handed her and tell the names of some.

She was discharged April 2, very much improved.

On May 17 she was reported by her home physician as being able to use her leg as well as ever. She had again become able to use her right hand in writing, but the fingers were a little stiff. Her vocabulary was fair, but not so good as before her illness. In all other respects she seemed perfectly well.

CASE 5.—For permission to publish this case we are indebted to Dr. S. Marx White, of Minneapolis, with whom one of us saw the patient in consultation, and to whom we are indebted for many of the notes referring to the case.

History.—Since 1901 the patient, a married man aged 54, had suffered from a bladder obstruction which annoyed and disturbed him to such a degree that, on April 6, 1902, a prostatectomy was done by Dr. J. E. Moore. This relieved the immediate difficulty. Up to about January 12 of the following year the patient was fairly well, but on this date the right epididymis was opened. On Oct. 17, 1905, the right epididymis was opened and removed.

During the year 1906 the patient was obliged to give up one-half of each day for rest. During this time also the urine became cloudy and was found to contain colon bacilli and staphylococci. There were no casts. Some small polypoid granulations sprang up at the margin of a blind pocket or partially closed sinus in the prostatic urethra, with intermittent obstruction to the flow of the urine. It was necessary occasionally to remove the granulations and irrigate the bladder.

In February, 1907, the obstruction became more pronounced and the use of the catheter began. In April, 1908, the patient went to St. Mary's Hospital in Rochester and Dr. C. H. Mayo performed a suprapubic cystotomy. The removal of a V-shaped portion of the fold of mucous membrane relieved the obstruction temporarily and there was some increase in the general strength. Hexamethylenamin (urotropin) and the catheter with bladder irrigation made up the treatment employed. Occasionally the suprapubic scar would open and drain for a few days. As an illustration it opened spontaneously June 30 and closed July 3, 1909. In spite of the treatment employed, however, the patient's general condition gradually failed. On July 6, 1909, he had a painless contraction of his left forearm, which lasted about an hour. The sinus opened July 19, 1909, and drained

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to Aug. 4, 1909, and on the last day mentioned, August 4, the patient complained of a peculiar numbness, which extended over his left hand, wrist and forearm, as if a tight glove were drawn over the parts. He did not speak much of this feeling, but would attempt to shake it off.

On August 5, his business partner sent an automobile to the house to take the patient for a ride and he seemed in every way normal mentally. In the afternoon of the same day, about 5 p. m., his wife sugested that he call up his partner and express his thanks. He was unable to recall the name until prompted, but telephoned in the usual manner and expressed his pleasure at the use of the car. A few moments later he turned to his wife and made an irrelevant and irrational remark, and within five minutes had a left-sided convulsion. Dr. White saw him at 6:30 p. m. The convulsions were being frequently repeated, and at the consultation at 8 p. m. the attacks were persistently evident in the left face and arm, while both legs were rigid.

No convulsions occurred except on the left side, but they continued at frequent intervals during the days and nights of August 6, 7, 8 and 9, and were uniform in character. There would develop suddenly a quick, violent, muscular movement of the left face and arm and occasionally of the left leg. The patient was able to talk at times, but his utterance was incoherent. His temperature began to rise with the onset of the convulsions and ranged from 99-8 to 103 F. On August 9, 1909, after a consultation including Drs. Moore, Tibbets, White and Jones, a diagnosis of acute septic encephalitis was made and an operation was advised for exploration and possible relief.

Operation.—The family having assumed the responsibility, on the following day, Dr. Moore removed a large osteoplastic flap from the right parietal region and exposed a large, very much congested and inflamed area in the motor cortex. No gross breaking down of the brain substance was noted, except in the center of the area where a softening process had begun. This area was incised and temporary drainage inserted.

Postoperative History.—There were no further severe convulsions, but slight and abortive seizures continued up to the time of death. The man became stuporous and died sixty hours after the operation. There was no autopsy.

The exact cause of the paralysis in the above cases it is quite impossible to give, and a consideration of the known facts concerning infantile hemiplegia does not serve to clarify the situation wholly.

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Hemiplegia due to thrombosis and embolism was established pathologically by Virchow in 1846. In 1876, Huguenin was able anatomically to distinguish clearly between encephalomalacia and encephalitis; and in 1884 Strümpell's article on "Acute Encephalitis of Children" brought out the fact that many of the cases of infantile hemiplegia are due to encephalitis of the motor area and this paper aroused special interest in the subject. In recent years much attention has been given to the histology and bacteriology of encephalitis, including especially in this country the valuable work of Councilman, Mallory and Wright, and of Southard, and his co-workers, Sims, Keene, Stratton and Richards. But up to the present it has been impossible to clinically distinguish the different groups of infantile hemiplegia according to their etiology (Southard), though the work of Oppenheim, Taylor and Southard will doubtless stimulate our activity and increase our capacity in this direction.

The deleterious influence of the diphtheria toxin on the nervous system has been long and widely known and both clinical and pathologic evidence combine to show that the nerves are much more subject to this influence than the ganglion cells of the cord and cortex. Peripheral paralysis occurs in diphtheria in as high as 5 to 15 per cent. of the cases, while out of 800 diphtheria patients in the Berlin Charity Hospital only three developed hemiplegia and out of Wallenberg's 190 cases of infantile hemiplegia only three were of diphtheritic origin, while of Osler's 120 cases not one was attributed to this cause. The histologic study of material from post-mortem cases of diptheria shows that changes in the ganglion cells are uncommon, and some authors record their entire absence. A few, however, insist on certain changes in the ganglion cells, though mostly of a light character, and in the small number of recorded post-mortem examinations the paralysis has usually been found to be due to embolus or thrombus. Taylor states that the causes of infantile hemiplegia in order of fre-

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quency are: acute encephalitis, thrombosis (arterial or venous), hemorrhage, and embolism. Very rarely a case has been reported in which the paralysis was believed to be partly of central and partly of peripheral origin.

Of the two cases of paralysis following diphtheria, here reported, there was undoubtedly a central lesion in both, but it may be that in Case 1 there was both a central and a peripheral lesion. Whether the cause was acute encephalitis, or one of the other conditions mentioned above, would have been difficult to determine at the time and is quite impossible with the history now at hand, though the prolongation of the symptoms previous to the development of the final and complete paralysis in both cases and the tendency of the symptoms to increase in intensity over a period of some days in one case would suggest encephalitis rather than one of the vascular conditions, which usually develop their symptoms comparatively rapidly.

Contrary to the opinion of the laity, paralysis in diphtheria is not due to the use of antitoxin, though the use of antitoxin does not appear to have lessened the number of cases of paralysis, either of central or peripheral origin.

Paralysis, either of central or of peripheral origin, in the course of pneumonia, is very infrequent. Thus Vanysek, in reporting a case of peripheral neuritis, states that neuritis and polyneuritis after croupous pneumonia is a very rare disease. Enouf reported a case of paralysis of his own and collected five others. In two of these cases, which came to autopsy, one had a meningeal hemorrhage and one a spinal cord softening. The other cases were of uncertain variety, including one case of paralysis of both legs and one arm. All four patients received after a duration of some hours to some weeks. Though there is no reason apparent why paralysis of central origin in pneumonia might not be due to true encephalitis, I do not know of a positive case in literature, except in connection with meningitis. There has

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been so little work done on the subject, however, that definite conclusions cannot yet be reached. As regards the nature of Case 4, in particular, no positive statement can be made. It was looked on previous to the operation as an instance of cerebral tumor or encephalitis. The operation and subsequent course make the diagnosis of tumor impossible and the diagnosis of encephalitis correspondingly more probable. In Case 5 the diagnosis of septic encephalitis seems justifiable.

513 Pillsbury Building.

ABSTRACT OF DISCUSSION

DR. M. L. PERRY, Parsons, Kan.: This question of infantile hemiplegia is one on which there has been division of opinion, especially as to the pathogenesis. When the paper on this subject was presented by Strümpell (in 1874, I believe), in which he took the position that the lesion in these cases was an encephalitis similar to or identical with the process found in anterior poliomyelitis, there were many, especially among the foreign men, who accepted the theory. It has not been accepted as fully in America and I think the weight of opinion among the men in this country has been until recently that these cases were of vascular origin. Dr. Southard, of Boston, has recently done some work that has thrown considerable light on this subject. He has found that the majority of these cases are due to an encephalitis, but the process is a different one from that found in the cord in anterior poliomyelitis. He has made rather extensive bacteriologic studies in such cases and has found that there is no one constant organism. I have myself been of the opinion that a large number of such cases were of vascular origin, until this work of Dr. Southard has convinced me that I have been mistaken. Viewed from a clinical standpoint the subject is of the greatest possible importance from the fact that a large number of such patients develop epilepsy later on. In a study of some nine hundred cases of epilepsy I have been much impressed with the frequency with which we find a history of attacks, similar to those described by the doctor in his paper, occurring as a precursor of epilepsy. In fact, as a result of a careful study of this subject I am convinced that the brain lesions causing infantile cerebral palsies may be ranked as one of the most important and frequent causes of epilepsy.

DR. SANGER BROWN, Chicago: It is a little doubtful whether these cases admit properly of a classification that would enable them all to be discussed together. According to the essayists the diphtheritic paralysis and encephalitis are adduced as the cause of the hemiplegia. I think the pathology of diphtheritic

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paralysis is pretty well defined to be a peripheral paralysis, and due to the effect of the toxin of diphtheria acting on the peripheral motor neurons, some being more sensitive than others, but in severe cases all being affected. There are various evidences, such as the usual impairment or loss of the reflexes in the parts concerned. Now I do not think in either of the two cases referred to by the essayists, even though signs of diphtheritic paralysis appeared before hemiplegia, that is, in the paralysis of the palate, or in the power of convergence, or the pupillary condition, even though that had occurred, and afterward hemiplegia occurred, it would be proper to regard the hemiplegia as being caused by the diphtheritic paralysis. It would be, I think, fair to assume that it was due to the general infection, as it might occur in any of the infectious diseases, and as Dr. Sachs has pointed out, or contended for very ably, the hemiplegia would be due to a vascular disturbance, due to the infectious disease, and not to the toxin acting on the peripheral neurons. I would strongly take the view that these two conditions were merely coincident, and I do not think it would be at all proper to regard the hemiplegia as a form of diphtheritic paralysis. Now, I think each case should be considered separately. The only other point that struck me was in the case of the child with the paralytic and sensory attacks in which there was no fever. The symptoms were described, and from the subsequent history I should strongly suspect interstitial sclerosis.

Dr. L. H. Mettler, Chicago: I would like to ask Dr. Perry or Dr. Hamilton how they differentiate, aside from the mere difference in location, between the pathology of acute hemorrhagic encephalitis and acute hemorrhagic anterior poliomyelitis. The theory propounded by Strümpell, wherein he classified certain forms of infantile encephalitis, nuclear inflammation, and anterior poliomyelitis as one and the same pathological process, differing only clinically by reason of the different localization of the lesion—a functional more than a pathologic differentiation—has always been more popular in Europe than in America. If Strümpell's condition is the true one, it seems to me that it offers us a distinct advantage in the practical consideration of these affections. Clinical differentiations between diseases, along easily observed lines of function, render the diagnosis and management of diseases easier and more accurate than when those differentiations depend on minute post-mortem pathologic findings. I, for one, would like to be more minutely informed on this matter. As I feel now, it is not the nature of the lesion so much as it is the location of it, that marks the distinction between these inflammatory troubles of the central nervous system, especially its gray matter. I think Starr takes emphatically the same position in his recent work on diseases of the nervous system. If these infantile palsies, cerebral, nuclear, and spinal, are absolutely dis-

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tinct and dissimilar disease processes, I think the differences, apart from mere location of the lesion, ought to be brought out more clearly by our teachers of pathology. Sarcoma of the brain and sarcoma of the cord are not regarded as essentially different diseases.

In this connection, while speaking of localization differences being made the basis of nosology in the absence of any real pathologic differences, I desire to speak of another matter not wholly irrelevant to the subject of the paper. Until the present time we have been talking a great deal about multiple neuritis, of the alcoholic and infectious types, as though it were a distinct, unique, and *sui generis* disease-process. Many pathologists now, however, are modifying their views, and are inclining to adopt more and more the teachings of Charcot, for which he long ago contended so earnestly and for which he received so much adverse criticism, namely, that these alcoholic intoxications and infections, when studied under the guise of multiple neuritis, represented mere localization differentiations and not essential diseases. Other organs were always more or less also involved, and the alcoholism or infection was the essential basis on which the cases in practice were to be classified. Present investigations show that in alcoholic polyneuritis, for instance, the degenerative process may be traced up through the nervous system to the very cortex. I cite this as a parallel instance somewhat to the question in hand, and to point out that we must not be too narrow in our conceptions of disease. We must be careful not to refer to clinical manifestations, which represent merely a functional localization, as special diseases.

Therefore, I would like to be informed wherein pathologically, not functionally, hemorrhagic polioencephalitis differs so much from hemorrhagic poliomyelitis as to warrant the consideration of these two maladies as wholly distinct and separate diseases. It is the end and aim of all science to generalize, if possible, to reduce to small groups many related phenomena, and to harmonize apparent discrepancies. It certainly is not the end and aim of science to merely enumerate a vast mass of minute and particular observations that may be closely interrelated.

Dr. W. A. Jones, Minneapolis: The object of this paper was to provoke a discussion as to the influence and effect of infections on the central nervous system. The description of the three supposedly diphtheritic cases means that infection is not only peripheral, but of central origin, and not infrequently destructive or inflammatory lesions are found in these so-called peripheral paralyses. If encephalitis and poliomyelitis are due to infections, I do not see how we can differentiate between these various diseases and their onset.

The two last cases reported, to my mind, are the most important, because they both show a central localized infective

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and inflammatory process in the cortex, and both cases, which were operated, seemed to confirm the work of Dr. Southard.

The third case, that of the child, was particularly interesting, but we were not able to determine the beginning of the infective process. The lesion was a characteristic one, if we can judge from gross appearances, and the inflammation involved mainly the cortex and did not extend into the white substance extensively. She made a prompt recovery from her peculiar epileptiform attacks and practically complete recovery from the hemiplegia following operation. The character of the epileptiform attacks in the third and fourth cases was distinctive in that the attacks were sudden, violent, repeated muscular contractions without paralysis between the seizures; that they were irritative and cortical I think is undisputed, particularly with our findings.

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SYPHILITIC DISEASE OF THE ARTERIES OF THE CENTRAL NERVOUS SYSTEM WITH DETAILED REPORT OF A CASE

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The importance of arteritis or endarteritis as a lesion of syphilis has become well appreciated, especially since the writings of Huebner, Baumgarten, Marchand, Rieder, Nonne, and others. Only recently, however, has sufficient emphasis been placed on the tendency of specific arteritis to appear shortly after primary infection, and on the relative frequency of isolated involvement of the arterial system as compared to the other rather more familiar forms of nervous syphilis implicating the meninges and surrounding structures of the central organ, etc.

As regards the time elapsing between the occurrence of the initial sore and the intervention of symptoms of arterial disease, the tendency of recent writers on this subject, especially Nonn, of Hamburg, is to insist on the very early development of arteritis after the primary infection. The latter author quotes a case in which symptoms of cerebral softening occurred even before the primary sore had healed.

Not only is arteritis now looked on as an early process of syphilis, but it is even held that this lesion in its pure form practically does not occur in very late syphilis, and that nearly all cases occur within three years after the chancre.

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Again it is necessary to recognize the frequency of arteritis as one of the grand diseases of the nervous system assignable to syphilis, because both treatment and prognosis are altogether determined by the nature of the processes in the nerve tissues associated with the arterial disease. Only by arriving at a proper conception of the lesions in such instances can the medical attendant properly interpret the phenomena occurring. It is rather probable that disappointment arising out of too sanguine expectations from the application of proper specific treatment, under the mistaken assumption that a gummatous or exudative process is being attacked, in instances of softening and hemorrhage in the central nervous organ, has been the occasion for physicians discarding the correct diagnosis of syphilitic disease, and consequently neglecting the effective agents of treatment subsequently.

It is unnecessary to dwell on the nature of the changes occurring in the arteries, because these are quite well understood. It is important only to recall that the process is practically always a proliferation of the intima with consequent encroachment on the lumen of the vessel and mechanical interference with the passage of the blood-current. And we do not have to wait for complete occlusion of the vessel in the expectation of symptoms, but a very important group of syphilitic nervous diseases is caused by mere diminution of the blood-supply to the tissues, giving symptoms of neurasthenia, transient pareses, and the like, not extending to the gravity of organic disease. In such instances the alterations in the arterial walls, especially in the smaller vessels, further interfere with the interchange of nutritive fluids, thus contributing to the so-called functional disturbances. As a matter of course, it is very important to interpret such symptoms correctly and early, for proper treatment is then most hopeful.

When actual hemorrhage, softening, or thrombosis occurs, the pathology and symptoms are in no wise pecul-

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iar to luetic arteritis, unless we regard the tendency of syphilis to select the branches of the middle cerebral artery as especially great. In my experience with a number of cases, I have found that the involvement of the arteries to the striate body, thalamus and the bulb especially frequent. The interesting lesions seen in the photographs of specimens from the case I am presenting, emphasize this. In this instance the lenticulo-striate group of arteries were almost all completely obliterated, leading to practically complete death of these structures on the right side and pronounced softening on the other.

I deem it somewhat foreign to the purpose of the present paper to more than refer to the fact that syphilis is the remote cause of aneurismal processes very late after the infection, operating in conjunction with agencies tending to raise the blood-pressure, such as atheroma, cardiac and renal diseases. This latter observation, however, serves in this connection to emphasize again the great tendency of syphilis to inflict wide-spread damage on the arterial tree.

I shall finish these few generalizations with reference to a case in which I was fortunate enough to follow the symptoms over a period of twenty months; the case finally coming to necropsy. Although I was not able to get the necropsy done in the best manner possible, and to prepare the specimens completely, I have succeeded in securing photographs and slides of two transverse sections which display the most pronounced lesions in a fairly satisfactory manner.

History.—The patient was a white man, aged 36, single, bartender by occupation, and came under observation March 26, 1908. Aside from the fact that the father was alcoholic, the family history was negative. The patient was exceptionally well as a child and during adolescence. In fact, there was no illness of consequence until he acquired syphilis. Beginning at the age of about 25, he led a very dissolute life, drank alcohol steadily and went on occasional week-end drunks; used

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tobacco and drank coffee to excess. During September, 1906, he contracted a venereal sore and had well-marked secondaries. Treatment was instituted and kept up in a desultory way for two years after appearance of secondary symptoms. There was seemingly no further manifestation of syphilitic activity until November, 1907, when the patient sustained a stroke of partial hemiplegia of the left side. The hemiplegic stroke was, however, preceded by dizziness and malaise for several weeks; and on treatment the weakness on the left side improved greatly, but never got entirely well. A short time before my observation, he became dull and confused, laughed and cried without cause, and showed other signs of disturbed mentalization.

Examination.—On first physical examination (March 26, 1908), the following observations were made: All deep reflexes were exaggerated, but those of the left arm and leg were most pronouncedly so; ankle clonus partially developed on left, exhausting after a few seconds; the left epigastric and hypogastric reflexes were absent; Babinski sign present on the left, Gordon sign on the right; Romberg test positive. It was impossible to test sensation carefully on account of the mental state, though there was obviously diminution of pain sense on the left side of the body. There was marked weakness of the left arm and leg. With the hands, 38 kilos on right and 12 on left, could be registered on the dynamometer. There was no sign of facial weakness on either side. Tongue deviated slightly to the left; pupils equal, round, active to light and accommodation. There was very slight swelling of the optic disks with obscuration of the edges and what appeared to be a mild toxic retinitis. The patient articulated awkwardly as if the tongue and lips were stiff on voluntary movement, and the voice was nasal. The soft palate sagged, but was observed to move on forced voluntary effort, not, however, on stimulation. The heart and other important viscera were normal; urine examination being likewise negative. The bowels were obstinately constipated and the tongue had a white coating. Control of the bladder was somewhat impaired, though this could not be determined well on account of the mental state. No hardening could be detected in the peripheral arteries. The mental state was one of universal reduction of all the psychic aptitudes. Patient was unable to tell where he was or anything about his situation. On attempting to answer questions, there was an overplay of the facial muscles of expression and articulation, and the effort would almost invariably end in a fit of crying. Words were enunciated in a clumsy way, amounting to dysarthria. The tendency to weeping obviously

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did not signify affective depression but the emotional instability seen in cases of gross lesions of the basal structures, especially the optic thalamus and striate bodies. The patient had no care for personal cleanliness. These and numerous other faults of behavior seemingly have their explanation in the mental state of bewilderment and negativism present. Specific treatment was given, but was so poorly tolerated that it had to be used very mildly. The digestive functions seemed to be in abeyance, constipation was obstinate and the general state grew worse.

Subsequent Course of Disease, May 4, 1908: General state somewhat worse than on last observation; patient required careful nursing, drooled saliva and refused food. Emotional instability continued; patient articulated badly; the gait was now somewhat more spastic; condition of the eye fundus remained the same; Babinski sign was present on both sides. Specific medication increased in quantity.

June 9, 1908: The general state was observed to be improved; patient would now articulate his words better, knew that he was in the hospital, took his food better and gained in weight; spasticity in extremities was much diminished; Babinski sign present on both sides; deep reflexes greatly plus; most pronounced on left.

June 16, 1908: Condition quite the same as on previous observation, possibly a bit better. Patient left the hospital.

During the month of October, 1908, patient was seen casually on the street and seemed almost normal. The gait was unimpaired and he asserted that he could use his extremities as well as ever. He felt strong and expressed intention of resuming his occupation. He was readmitted Aug. 15, 1909, having become practically helpless in all four extremities.

October 2, 1909, the following observations were made: Patient's condition was worse than when first seen. He cried almost continually; signs of the old left hemiplegia were again prominent, and the right side was also weak and spastic; the arms were stiff and awkward on voluntary movement; pronounced weakness in both arms and legs, but most pronounced in left; deep reflexes greatly exaggerated on the left, less so on the right; sustained ankle-clonus on the left; Babinski sign present on both sides. Sensation could not be tested on account of mental state. No athetoid movements or tremors present. The mental state was the same in kind as that seen on first observation, though of greater depth. The patient lay quietly and discharged the functions of his body without concern, requiring to be fed like a child. Articu-

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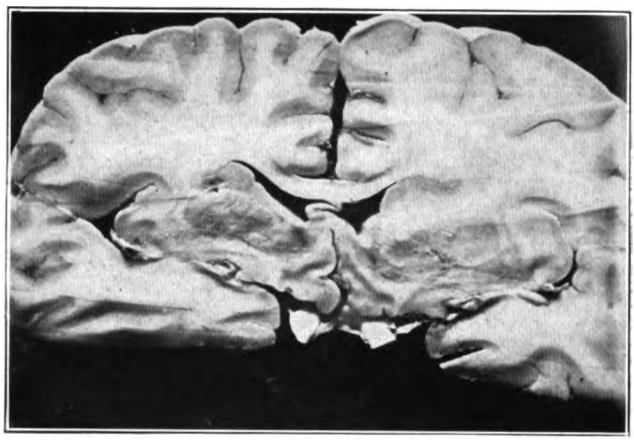

Fig. 1.—Syphilitic endarteritis.

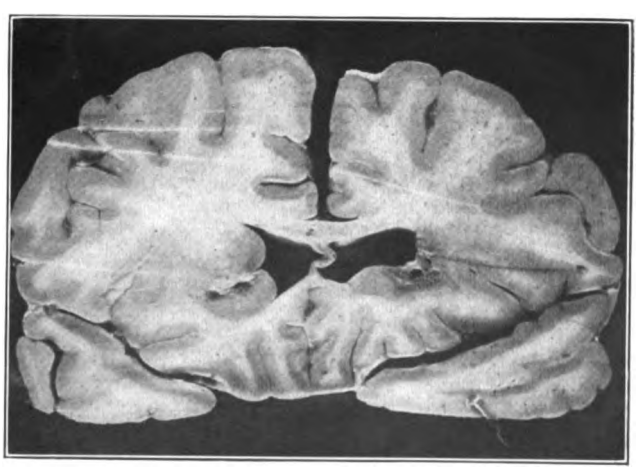

Fig. 2.—Syphilitic endarteritis.

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UNIV. OF MICH.

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lation was scarcely intelligible, and he had great difficulty in swallowing. Specific treatment was again instituted, but totally disagreed and had to be withdrawn.

Nov. 28, 1909: Patient was bedridden and could scarcely swallow food from involvement of deglutitory muscles. The mental state was one of negativism, due to the profound reduction of all cerebral functions. He still continued to cry when aroused. Babinski sign was present on both sides; sustained ankle-clonus on left. The optic disks remained partially obscured as noted on previous observations. The patient moved his extremities in an awkward, ataxic, spastic manner; was obstinately constipated and abdomen was retracted. Patient was unable to swallow and seemed not to assimilate nourishment given by stomach-tube.

He died December, 1909, from inanition.

Clinical Diagnosis.—Syphilitic endarteritis of the central nervous system, with focus of hemorrhage or softening at first in the right internal capsule; later similar involvement of the left capsule. Probably multiple small foci about the hemispheres from rupture or obliteration of the smaller terminal vessels.

Post-mortem Findings.—There were found slight universal thickening of the dura, and wide-spread opacities of the pia with cystic formations in places. The walls of the large vessels at the base were observed to be thickened, especially the middle cerebral and its branches. Areas of softening were seen in the region of the two striate bodies, most marked on the right, where the lesion appeared older and involved the thalamus and practically all structures in the lenticulo-striate region, apparently destroying the majority of the fibers of the capsule. These were the two chief gross lesions, but numerous minute foci of necrobiosis were seen in the cortex and subcortical substance of the hemispheres.

Transverse sections of the spinal cord showed the left half of this organ to be distinctly less in volume than the right. The pyramidal tracts were seen to be greatly atrophied on naked eye inspection, apparently from secondary degeneration alone.

On account of the difficulties under which our observations were made, no Wassermann reaction could be done nor search for spirochetes made; but the clinical history and appearances of the lesions found were so overwhelmingly in favor of syphilis as to leave no reasonable doubt.

I realize the great interest that might have attached to a more detailed observation of the symptoms in the

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case and the exhibition of microscopic sections of the brain and cord in further exposé of the beautiful pathology present. Lack of opportunity prevented my doing the former. I hope to complete the pathological work later.

I have the pleasure to express my thanks to Drs. Daspit, Duval, and Couret, who have so kindly assisted me in securing and preparing the specimens.

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ABSTRACT OF DISCUSSION

DR. WILLIAM W. GRAVES, St. Louis: Dr. Hummel referred to the frequency of early arterial degenerations as a manifestation of syphilis, especially as pointed out by Nonne in recent years. This great clinical fact is one which is not appreciated. Early arterial degeneration after a syphilitic infection, no matter how carefully treated, is the one preeminent clinical fact on which one may oftentimes hinge a provisional diagnosis. Signs of arterial degeneration, occurring in a man before his time, and one being able to exclude metallic and other poisons as the cause, are in the majority of cases due to a former syphilitic infection. I base this conclusion not on that finding alone, but there are other clinical findings demonstrable in cases of syphilis two or more years after the infection, which are almost as constant as the arterial changes.

During the last few years I have given much attention to syphilis of the nervous system and post syphilitic conditions, and I have made careful studies not only of the arterial systems of syphilitics, but also of their nervous systems; and from these studies it may be stated, though I may be considered radical when I do state it, that there are but few cases, however carefully we may treat them, who two years after a syphilitic infection will not show organic changes as demonstrable by a clinical consideration of the nervous system. Indeed during the secondary manifestations of syphilis in some cases which I have had the opportunity of studying, I have found distinct sensory loss, especially to pin pricks. This loss may and probably does remain throughout the life of the individual. A syphilitic may lead a healthful life, but he reacts badly. The individual who has had a syphilitic infection must be regarded as a blighted individual, reacting poorly to stresses and strains. Such individuals when studied, in addition to cardiovascular changes, show in the majority of cases changes in the oculo-pupillary apparatus and in the nervous system.

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Dr. George A. Moleen, Denver: I have been greatly pleased with the case report that has just been made, and I believe that this condition is too often neglected by reason of its frequent occurrence. The first phase that appeals to me in this paper is the evidence of neglected syphilis which has been the cause of the arterial degeneration. I have seen quite a number of cases of cerebral arterial degeneration in Colorado, and almost invariably the history of the syphilitic infection carries with it an incompleted treatment, or a treatment extending usually not over two or three months. The length of time of specific treatment, I think, is often in direct relation to the occurrence of an arteritis. I have seen in one instance the occurrence of a syphilitic arteritis within three months of the initial sore. In other cases they have gone, as has been the experience of others, as long as fifteen or twenty or more years. The mental confusion in the case just cited, I take it, is an expression of the general toxic effect of the syphilitic virus, which is further evidenced by the neuritis and the retinitis, and these are not commonly, if ever, present in pure arterial lesions. I do not believe that many such cases should occur if the proper mercurial treatment were established and continued, and particularly continued when once established. I think with Osler that it is lamentable that we should find arterial lesions in specific cases like these, which are so amenable to mercurial treatment.

Dr. Theodore Diller, Pittsburg: There is a good deal of thickening of arteries, a good deal of arteriosclerosis, that is not syphilitic. For instance, I very often find thickening of arteries in boys with dementia præcox, and I believe that there is a good deal in the inheritance of bad arteries, arteries which degenerate early, and I feel that we ought to be not too hasty in concluding that thickened arteries indicate specific disease, even in young persons.

Dr. E. M. Hummel, New Orleans: Fourteen months elapsed between the time of the initial sore and the stroke of hemiplegia which this man suffered, which in the light of subsequent developments was undoubtedly assignable to softening in the right capsule due to obliterating endarteritis. I wish to insist again on the early occurrence of signs of specific endarteritis, dating from the time of the initial sore. Dr. Nonne in his comprehensive treatise, "*Syphilis und Nerven-System*," insists on this repeatedly, and comes to the conclusion that syphilitic processes of this kind always occur practically within three years of the time of the infection. I did not wish to be understood as confusing specific endarteritis with arteritis of other causation, and I quite agree with Dr. Diller that there are numbers of other kinds of endarteritis, but I think, everything considered, there is no doubting the specific character of the arteritic process in this case. There is one point

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that I should like to mention further in connection with the case, and that is that it is noted in the clinical symptoms that this man underwent great improvement under specific treatment and got practically well. The explanation of that is interesting. It is probable that there was a large exudate around the site of the original softening, and that it was absorbed under specific treatment. One thing further in this connection and of interest here is that when a person under the age of 40 sustains a stroke of hemiplegia or any other sign of gross focal, brain or spinal cord trouble, coming on suddenly, indicating either hemorrhage or softening, the presumption is very strong that such an accident at so early an age indicates the presence of arterial disease of specific causation and the case ought not to be deprived of a therapeutic trial with antisyphilitic agents.

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THE HEMIANOPTIC PRISM-PHENOMENON OF WILBRAND

ALFRED SAENGER, M.D.
HAMBURG, GERMANY

By the method devised by Wilbrand we are able to differentiate between tract hemianopsia and cortical hemianopsia.

You all know the hemianoptic pupil reaction described by Wernicke; but all investigators are of the opinion that this symptom is very difficult to see on account of the dispersion of light in the eye. The macula is irritated and pupillary reaction appears in consequence.

The new method of Wilbrand is best described as follows: The patient is requested to fix a white point on a large black plane and direct his attention to it. In this way all other impressions on the retina are excluded. The patient takes his place at a distance of thirty to fifty centimeters just in front of the point. Suddenly two prisms of equal degree (e. g. 15°) are brought before both eyes, the apex turned to the hemianoptic defect. By this means the white point is directed into that side of the retina which is not perceptive. The basis of the prisms must be parallel.

If a cortical hemianopsia exists, the patient will quickly change the direction of sight and move the eyes so that the fovea is directed to the object. This proves that there exists a reflex independent of the cerebrum, and, on the other hand, that the optical route between retina and corpus geniculatum laterale is free.

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If the reflex way is interrupted by a lesion of the tractus opticus, the eyes under the prisms make no movement to fix the object, but remain quiet.

ABSTRACT OF DISCUSSION

Dr. William W. Graves, St. Louis: I am able out of my own personal observation to present two cases of hemianopsia for Dr. Saenger's consideration; the first, a young lady, 17 years, who about four years ago began having epileptic seizures. She was treated for epilepsy about two years without success. She consulted Dr. J. F. Shoemaker for failing vision, who found decided choked disc, and he referred the case to me. The examination of her nervous system otherwise indicated the presence of a tumor in the vicinity of the optic chiasm. Her rapidly failing vision caused me to advise an immediate operation. She was decompressed by Dr. V. P. Blair according to Cushing's subtemporal operation, and one may see the point of decompression. She has been improved in every way. Uncontrollable headaches, dizziness and sleepy spells have entirely disappeared. She still has spells of headaches, and when severe, we relieve her with lumbar puncture. The visual fields in the beginning showed a decided contraction and interlacing, as pointed out by Cushing and Bordley; but they have shown a very decided variability.

Her vision, which was really quite defective, so that she could no longer read large print, has become normal since the subtemporal decompression and is now about 18/15 in both eyes. But she still has very narrow fields. Her fields indicate that the lesion is in the optic tract or about the optic chiasm, a little more upon the left than the right. We feel that we have been able to exclude anything like a lesion of the optic radiations themselves or a lesion about the calcarine fissure. So that this then would be a splendid case for the demonstration of the sign mentioned by Dr. Saenger and this case is presented to Dr. Saenger as a case of lesion probably involving the left optic tract or the left part of the chiasm.

The second case is that of a young man, aged 22, who was hurled to the earth in a football game and remained unconscious for three months. One of the first observations he made was that all objects coming into his fields of vision from the right side were not seen until they were passed his nose, and this defect has remained practically the same. I saw him first in November, 1909, about a year after the accident. Not only had he a right homonymous hemianopsia but double-sided ataxia, bradyphasia, double-sided toe reflexes, absent abdominal reflexes, and interesting sensory

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phenomena, the most prominent of which was his inability to distinguish between weights, as between one ounce in his right hand and 8 ounces in the left hand, both weighing the same. I may remark that amnesia has been very noticeable. While he recognized schoolmates six months later he could not recall their names, so that he had a true defect in that regard. He still shows disturbance in sensation, particularly about the distal portions of all extremities, more marked on the right than on the left. Careful palpation of the skull revealed above the occipital protuberance on both sides, distinct depressions. Those depressions were also verified by an *x*-ray, which showed slight lineal fractures. There is shown in the charts a more or less complete right hemianopsia. The lesion here is assumed to be about the left calcarine fissure.

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INDEX

PAGE

Allison, N.: discussion on anterior poliomyelitis..... 217
Amyotonia congenita; paper by A. L. Skoog..... 137
Bailey, Pearce: paper on the dependence of neurology on internal medicine 25
Bassoe, P.: discussion on anterior poliomyelitis..... 213
Bordley, J., Jr.: discussion on choked disc and brain tumor... 167
Briggs, L. V.: discussion on neurologic hospitals..... 49
Brown, S.: discussion on infantile hemiplegia..... 248
Chesley, A. J.: paper on pathology and bacteriology of acute anterior poliomyelitis 170
Church, Archibald: paper on nervous and mental disturbances of the male climacteric 129
Discussion on anterior poliomyelitis..... 211
Climacteric, male, nervous and mental disturbances of; paper by A. Church 129
Collins, Joseph: paper on the dependence of neurology on internal medicine 25
Discussion on same 48
Crothers, T. D.: discussion on neurologic nospitals..... 43
Dana, C. L.: discussion on brain tumor..... 95
Discussion on neurologic hospitals..... 43
Discussion on tumor of spinal cord..... 234
Diller, Theodore: paper on institutional treatment of nervous and mental diseases 15
Discussion on brain tumors..... 96
Discussion on syphilitic arteritis 259
Discussion on tumor of spinal cord..... 232
Discussion on work cure and gymnastics..... 127
Disc, choked, in its relation to cerebral tumor and trephining; paper by A. Saenger 155
Discussion 166
Emerson, C. P.: discussion on work-cure and gymnastics..... 125
Fry, F. R.: discussion on anterior poliomyelitis..... 218
Discussion on neurologic hospitals..... 45
Discussion on work-cure and gymnastics..... 124
Gaub, O. O.: paper on intradural tumor of the spinal cord.... 222
Graves, W. W.: discussion on syphilitic arteritis 258
Discussion on hemianopsia 262
Grinker, Julius: paper on tumor in the cerebello-pontile angle. 81
Discussion on same 98
Discussion on choked disc and brain tumor..... 166
Discussion on tumor of spinal cord..... 233
Hall, G. W.: discussion on brain tumors..... 95
Hall, Herbert J.: paper on manual work in the treatment of the functional nervous diseases..... 108
Discussion on same 128
Hamilton, Arthur S.: paper on hemiplegia following acute infections 235
Discussion on anterior poliomyelitis 216

Digitized by Google

PAGE
Hecht, D'O.: discussion on brain tumors..... 96
Hemianoptic prism-phenomenon of Wilbrand; paper by A. Saenger 261
Discussion 262
Hemiplegia following acute infections; paper by W. A. Jones and A. S. Hamilton..... 235
Discussion 248
Hoppe, Herman H.: paper on the topographic diagnosis of subtentorial brain tumors..... 50
Discussion on neurologic hospitals..... 47
Hughes, C. H.: discussion on anterior poliomyelitis..... 219
Discussion on brain tumors..... 94
Hummel, E. M.: paper on syphilitic arteritis of the central nervous system 252
Discussion on same 259
Inebriate, state care of; paper by I. H. Neff..... 34
Discussion 42
Jones, W. A.: paper on hemiplegia following acute infections. 235
Discussion on same..... 250
Kramer, Simon Pendleton: paper on brain tumor of psychomotor area 76
Langdon, Frank Warren: paper on brain tumor of psychomotor area 76
Mayer, E. E.: paper on intradural tumor of the spinal cord.... 222
McCready, E. Bosworth: paper on the relation of stuttering to amusia 148
Mettler, L. H.: discussion on anterior poliomyelitis 212
Discussion on infantile hemiplegia..... 249
Discussion on work-cure and gymnastics..... 126
Moleen, G. A.: discussion on syphilitic arteritis..... 259
Narcotics, treatment of acute ailments in persons addicted to; paper by G. E. Pettey..... 99
Neff, Irwin H.: paper on state care of the inebriate..... 34
Discussion on same..... 49
Nervous and mental diseases, institutional treatment of; paper by T. Diller..... 15
Nervous diseases, functional, manual work in treatment of; paper by H. J. Hall..... 108
Discussion 123
Nervous diseases, gymnastics and massage in the treatment of; paper by H. Sneve..... 115
Discussion 123
Neurology, dependence of, on internal medicine; paper by J. Collins and P. Bailey..... 25
Discussion 42
Norbury, F. P.: discussion on neurologic hospitals..... 44
Paralysis, infantile spinal, surgical treatment of; paper by D. Silver 196
Discussion 211
Parsons, J. H.: discussion on choked disc and brain tumor.. 166
Patrick, H. T.: discussion on neurologic hospitals..... 45
Perry, M. L.: discussion on infantile hemiplegia..... 248
Pettey, George E.: paper on treatment of acute ailments in persons addicted to narcotics 99

Digitized by Google

PAGE

Poliomyelitis, acute anterior, pathology and bacteriology of; paper by H. E. Robertson and A. J. Chesley..... 170
Discussion 211
Punton, J.: discussion on work-cure and gymnastics..... 124
Robertson, H. E.: paper on pathology and bacteriology of acute anterior poliomyelitis 170
Discussion on same 220
Saenger, Alfred: paper on choked disc and cerebral tumor and trephining 155
Discussion on same..... 168
Paper on hemianoptic prism-phenomenon of Wilbrand. 261
Discussion on neurologic hospitals..... 42
Silver, D.: paper on the surgical treatment of infantile spinal paralysis 196
Discussion on same..... 220
Skoog, A. L.: paper on amyotonia congenita..... 137
Discussion on anterior poliomyelitis..... 215
Sneve, Haldor: paper on gymnastics and massage in the treatment of nervous diseases..... 115
Discussion on same..... 128
Discussion on neurologic hospitals..... 46
Spinal cord, intradural tumor of, with operation; paper by E. E. Mayer and O. C. Gaub..... 222
Discussion 232
Stuttering, relation of to amusia; paper by E. B. McCready... 148
Syphilitic disease of the arteries of the central nervous system; paper by E. M. Hummel..... 252
Discussion 258
Tumor, brain, of psychomotor area; paper by F. W. Langdon and S. P. Kramer 76
Discussion 94
Tumor, cerebral, and trephining, choked disc in its relation to; paper by A. Saenger..... 155
Discussion 166
Tumor, intradural, of the spinal cord, with operation; paper by E. E. Mayer and O. C. Gaub..... 222
Discussion 232
Tumor in the cerebello-pontile angle; paper by J. Grinker.... 81
Discussion 94
Tumors, subtentorial brain, topographic diagnosis of; paper by H. H. Hoppe..... 50
Discussion 94
Woodson, C. R.: discussion on anterior poliomyelitis..... 219
Discussion on work-cure and gymnastics..... 123
Zenner, P.: discussion on work-cure and gymnastics..... 125

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